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CONGENITAL INSUFFICIENCY OF THE PALATE*

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PHILADELPHIA

He who knows the past can best interpret the present

English: Congenital insufficiency of the palate
Congenital shortening of the palate
Submucous cleft palate

French: Brièveté vélo-palatine
Insuffisance vélo-palatine

German: Submukoese Spaltung des harten Gaumens
Fissura ossea occulta
Fissura muscularis occulta

Italian: Insufficienza velo-palatina

Congenital insufficiency of the palate is a condition in which the velum, assisted by the superior constrictor muscle of the pharynx, fails to produce sphincter-like closure between the nasopharynx and the oropharynx, a condition essential for the production of normal speech. While congenital insufficiency of the palate has probably existed from the beginning of the human race, no reference to it occurs in the literature until 1825.

Roux,[1] in 1825, first called the attention of the medical profession to congenital insufficiency of the palate. He stated in his paper that in 1823 he was consulted by a young girl who nasalized so badly that her speech was unintelligible. He described in this girl a cleft of the posterior portion of the velum, and stated that there was faulty union of the osseous tissue of the hard palate under an intact mucosa. As far as I have been able to ascertain by an exhaustive study of the literature, Roux was the first to describe this variety of submucous cleft palate.

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* The artist's drawings which appear in this essay were made by Mr. Erwin F. Faber of Philadelphia.

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1. Roux, Joseph Philibert: Mémoires sur staphylorrhaphie, Paris, J.-S. Chaudé, 1825, p. 84.

Demarquay,[2] in 1846, exhibited a dissection in which there was a cleft of the velum and the bony palate, the cleft in the latter being filled with fibrous tissue; the mucosa was intact.

In 1862, Passavant[3] described the absence of a median triangular portion of the posterior part of the bony palate. The mucosa over this portion was intact. The patient also had a cleft of the velum and the alveolar border. Passavant stated that healing occurred in the velum at the age of 15 months.

Langenbeck,[4] in 1864, differentiated three forms of congenital anomalies of the palate in which there was absence of bony tissue underneath the unaffected mucosa. He described the following varieties:

1. Cases of cleft of the velum in which the bony palate is almost entirely absent, although the mucosal covering is present.
2. Cases of cleft of the velum and hard palate in which the fissure in the bony palate is more extensive than in the soft parts covering the palatine vault.
3. Cases of cleft of the velum associated with a fine split either in the midline or to either side of the vomer, with the mucosa intact.

Notta,[5] in 1869, reported a case of a girl, aged 11 years, in whom the uvula was cleft. Under the intact mucosa there was a triangular breach in the substance of the hard palate, extending from the incisive fossa and ending posteriorly with its base in line with the last molar tooth. The patient spoke with a distinct nasal twang.

In 1869, the term "brièveté de la voûte palatine" was introduced by Trélat.[6] Trélat attributed the nasal intonation to the anteroposterior shortening and lateral narrowing of the hard palate. He also recognized the association of anteroposterior brevity of the bony palate and described the notching of the palate bones toward the velum.

Gayraud,[7] in 1884, called the attention of the medical profession to the fact that this type of congenital anomaly, namely, anteroposterior shortening of the palate, was much more common than was hitherto supposed, and he felt that it was the chief factor in causing nasal speech. Lermoyez,[8] in 1892, introduced the term "insuffisance vélo-palatine," and in a lucid and detailed treatise described twelve cases.

The first one to call attention to the condition in America was Mears[9] of Philadelphia, who in 1893 reported a case in a young lady

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2. Demarquay: Bull Soc. anat. de Paris **21**:11, 1846.
3. Passavant, Gustav: Arch. d. Heil. **3**:334, 1862; Arch. f. klin. Chir. **6**: 333 and 587, 1865.
4. Langenbeck: Arch. f. klin. Chir. **5**:7, 1864.
5. Notta: Bull. Soc. de chir. de Paris (1869) **10**:419, 1870.
6. Trélat, U.: Bull. Soc. de chir. de Paris (1869) **10**:402, 1870.
7. Gayraud, B.: Dictionnaire encyclopédique des sciences médicale, Paris, Masson et fils, 1884, vol. 19, pp. 698 and 702.
8. Lermoyez, Marcel: Ann. d. mal. de l'oreille, du larynx **13**:161, 1892.
9. Mears, J. Ewing: Southern M. Rec. **24**:59, 1894; Clefts of the Hard and Soft Palate, Philadelphia, 1893.

with marked shortening of the velum and a pronounced nasal and gutteral speech.

Botey [10] of Barcelona devised an operation in 1907 for the correction of this deformity. Further details of this operation will be discussed later in the body of this paper.

The term "submucous cleft palate" was introduced into the literature in 1910 by Kelly [11] of London, who made a thorough investigation of this congenital malformation and published the first complete and systematic English treatise dealing with this subject.

Seeman [12] of Prague contributed valuable papers on the symptomatology and pathology of submucous clefts of the hard palate.

In 1927, Limberg [13] of Leningrad introduced the terms "fissura ossea occulta," by which he meant submucous cleft of the hard palate, and "fissura muscularis occulta," by which he meant submucous cleft of the soft palate.

That the subject of insufficiency of the palate has attracted the attention of numerous observers is evidenced by the fact that mention of this condition has been made by every important worker in the field of cleft palate, among whom are mentioned:

Simon,[14] Ehrmann,[15] Wolff,[16] Fränkel,[17] Kayser,[18] Castex,[19] Fröschels,[20] Egger,[21] Neugenbauer,[22] Bonne,[23] Garel,[23] Fein,[24] Gutz-

10. Botey, Ricardo: Rev. de cien. méd. **33**:433, 1907; Arch. Internat. de laryng. **24**:392, 1908.

11. Kelly, A. Brown: J. Laryng. & Otol. **25**:281 and 342, 1910.

12. Seeman, Milo: Berl. klin. Wchnschr. **56**:377, 1919; Monatschr. f. Ohrenh. u. Laryngo-Rhinol. **53**:510, 1919; Arch. Internat. de laryngol., oto-rhinol. et broncho-oesoph. **3**:388, 1924; Cas. lék. česk. **62**:461, 1923; Zentralorgan f. d. ges. Chir. **23**:457, 1923; Cas. lék. česk. **61**:811, 1922; Zentralorgan f. d. ges. Chir. **21**:179, 1923.

13. Limberg, Alexander: Zentralbl. f. Chir. **54**:1745, 1927; J. Am. Dent. A. **14**:1113, 1927.

14. Simon, Gustav: Greifswalder medizinische Beiträge, Danzig, 1864, vol. 2, p. 169; Vrtljschr. f. d. prakt. Heilk. **1**:1, 1867; Mitteilungen aus der chirurgischen Klinik de Rostocken Krankenhauses, 1861-1865, Prag, Reichenecker, 1868, p. 80.

15. Ehrmann, J.: Gaz. méd. de Strasb., 1880, p. 118.

16. Wolff, Julius: Verhandl. d. deutsch. Gesellsch. f. Chir. **14**:387, 1885; **23**: 461, 1894.

17. Fränkel: Berl. klin. Wchnschr. **19**:582, 1882.

18. Kayser, B.: Deutsche med. Wchnschr., 1891, p. 825.

19. Castex, A.: Ann. d. mal. de l'oreille, du larynx **19**:415, 1893.

20. Fröschels, Emil: Wien. med. Wchnschr. **61**:2537, 1911.

21. Egger, L.: Ann. d. mal. de l'oreille, du larynx **22**:365, 1986.

22. Neugenbauer, Bruno: Ueber Messungen des weichen Gaumens mit Darstellung einer neuen Messungemethode, Inaug. Diss., Koeningsberg, 1896.

23. Bonne, André: De la Rhinolalie, Thése de Lyon, 1897, no. 92.

24. Fein, Johann: Wien. klin. Wchnschr. **12**:76, 1899; **9**:982, 1896.

mann,[25] Gersuny,[26] Lannois,[27] Chiari,[28] Helleu,[29] Philip,[30] Rouvillois,[31] Brown, [32] Stimson,[33] Foucou,[34] Blair,[35] Roberts,[36] Moure,[37] Grünberg,[38] Peter,[39] Kenyon,[40] Stern,[41] Imhofer,[42], Franke,[43] Bumba,[44] Apert and Bigot,[45] Rabattu,[46] Dreyfuss,[47] Nadoleczny,[48] Davis,[49] Ivy,[50] Mościsker [51] and Kindler.[52]

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25. Gutzmann, Hermann: Berl. klin. Wchnschr. **36**:813, 1899; Physiologie der Stimme und Sprache, Braunschweig, Vieweg, 1928; Von den verschiedenen Formen des Naeselns, Halle, C. Marhold, 1901.

26. Gersuny, R.: Ztschr. f. Heilk. **21**:199, 1900.

27. Lannois, M.: Rev. hebd. de laryngol., d'otol. et de rhinol. **21**:177, 1901.

28. Chiari, Ottokar: Die Krankheiten der Nase, Leipzig, F. Deuticke, 1903, vol. 2, p. 202.

29. Helleu, Maxime: Le pronostic des urano-staphylorraphies pour fissures congénitales, Thése no. 313, Paris, 1904.

30. Philip: Ann. d. mal. de l'oreille, du larynx **30**:597, 1904.

31. Rouvillois: Rev. hebd. de laryngol., d'otol. et de rhinol. **28**:753, 1908.

32. Brown, G. V. I.: Speech Results of Cleft Palate Operations, J. A. M. A. **50**:342 (Feb. 1) 1908; Dental Cosmos **50**:123, 1908; Lancet-Clinic **105**:256, 1911.

33. Stimson, G. W.: Congenital Insufficiency of the Palate, J. A. M. A. **52**: 559 (Feb. 13) 1909.

34. Foucou: Rev. méd. de la Suisse Rom. **30**:649, 1910.

35. Blair, V. P.: Tr. Surg. & Gynec. A. **23**:479, 1911; Surg. Gynec. Obst. **12**:289, 1912.

36. Roberts, John B.: Internat. Clin. **4**:220, 1918; Pennsylvania M. J., 1920-1921, p. 64.

37. Moure, E. J.: J. de méd. de Bordeux **84**:639, 1913.

38. Grünberg, Karl: Die Morphologie der Missbildungen des Menschen und der Tierre, part 3, Jena, Gustav Fischer, 1913, p. 113, chapter 4.

39. Peter, Karl: Atlas der Entwicklung der Nase und des Gaumens beim Menschen mit Einschluss der Entwicklungestoerungen, Jena, Gustav Fischer, 1913, p. 112.

40. Kenyon, Elmer L.: Ann. Otol. Rhin. & Laryng. **22**:1110, 1913; **34**:887, 1925.

41. Stern, Hugo: Monatschr. f. Ohrenh. u. Laryngo-Rhinol. **53**:577, 1919; **54**:255, 1920; Arch. f. Laryngol. u. Rhinol. **34**:95, 1921.

42. Imhofer, R.: Ztschr. f. ang. Anat. u. Konstitutionslehre **5**:146, 1920.

43. Franke, Gustav: Ueber Wachstum und Verildungen des Kiefers und der Nasenscheidewand, Leipzig, Kabitzch, 1921, p. 68.

44. Bumba, Josef: Monatschr. f. Ohrenh. u. Laryngo-Rhinol. **56**:225, 1922.

45. Apert and Bigot: Bull. Soc. de pédiat. **20**:28, 1922.

46. Rabattu, J.: Rev. de laryngol., d'otol. et de rhinol. **44**:859, 1923.

47. Dreyfuss: Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:253, 1925.

48. Nadoleczny, Max: Kurzes Lehrbuch der Sprach- und Stimmeheilkunde mit besonderer Berueksichtigung des Kindesalters, Leipzig, F. C. W. Vogel, 1926, p. 71.

49. Davis, Warren B.: Ann. Surg. **87**:536 (April) 1928.

50. Ivy, Robert Henry: Personal communication to the author, January, 1927.

51. Mościsker, E.: Wien. med. Wchnschr. **78**:961, 1928.

52. Kindler, Wenner: Passow-Schaefer Beitr. z. Anat., Physiol., Path. u. Therap. d. Ohres. **27**:187, 1928.

VARIETIES

Congenital insufficiency of the palate may vary in accordance with the following forms:

1. Cases in which the entire palate is normal in appearance, but in which the velum is unable to approximate the pharyngeal wall because of anteroposterior shortening of the hard palate and the velum.
2. Cases in which the velum is normal in length and in which the hard palate is short.
3. Cases in which the hard palate is normal and the velum shorter than normal.
4. Cases in which the hard palate is normal in length and outline but in which there is a submucous cleft of the velum.
5. Cases in which the velum is normal in appearance and length and in which there is a submucous cleft extending into the hard palate.
6. Cases of insufficiency of the palate which occur after successful cleft palate operations, the velum being too short to reach the posterior pharyngeal wall.

ETIOLOGY

At the present time, the etiology of congenital insufficiency of the palate is unknown. The same conditions which cause other types of cleft palate, namely, an arrest in development due to some abnormal disturbance,[53] may result in congenital insufficiency. Just what the factors are that produce these deviations, one cannot say. It is known that heredity plays an important rôle, for defects may be handed down from generation to generation. Any type of defect may repeat itself in the offspring, and it is well known that persons with congenital anomalies of the palate frequently present malformations in other parts of the body. It is interesting to note the explanation offered for congenital deformities of the palate.

Fortunius Licetus,[54] in his writings published in the early part of the 17th century, recorded the accepted opinions of his time, regarding many of them as absurd. Among these were some of the widely prevalent Aristotelian views. Licetus attributed these anomalies to diseases of the fetus, maternal impressions, superfecudation and nutritional disturbances affecting the developing fetus.

Pierre Sylvian Régis,[54] in 1690, first called attention to the important rôle played by heredity in predisposing the transmission of these deformities. His views were supported by Winslow, Haller, Meckel and others.

Geoffrey St. Hilaire [55] proposed that mechanical influences were the main factors that produced these anomalies. Panum and Dreste

53. Rokitansky, Carl: A Manual of Pathological Anatomy, Philadelphia, 1855, vol. 2, p. 17.

54. Quoted by Tirifahy, S.: J. de méd. e chir. et de pharmacol. **37**:335 and 422, 1863.

55. Quoted by Kramer, Josephy: Ueber Wolfsrachen und frühzeitige Uranoplastik, Inaug. Diss., Kiel, 1900.

attempted to support Hilaire's views by experimentation. Panum produced deformities in chickens by painting the eggs with varnish, and Dreste accomplished the same by placing the incubating eggs on one end.

Vrolik and Nicati [54] also favored the mechanical theory, holding that the cleft palate was the result of increased development of the tongue which interposed between the palatal plates, preventing them from uniting in the midline. His corroborated this theory and added that he felt that the upward pressure of the fist of the fetus on the mandible was instrumental in wedging the tongue between the palatal plates. Panum later referred to the influence of amniotic adhesions in producing deformities of the palate.

Ahlfeld [56] attempted to explain the presence of clefts by calling attention to the fact that hydrocephalus was a frequent factor in these cases. He felt that the widening of the base of the anterior fossa of the skull in cases of hydrocephalus prevented the palatal plates from meeting in the midline. Fein,[24] an ardent supporter of the mechanical theory, called attention to the presence of adenoids in cases of cleft palate, attributing their mechanical influence as a cause for the production of these deformities. The association of adenoids with cleft palate or submucous cleft palate is certainly not a factor in producing the deformity, since adenoids do not appear in the fetus until the third month of intra-uterine life, a period at which the palate is completely formed.

Warnekros,[57] demonstrating by means of the x-rays the presence of supernumerary teeth in cases of cleft palate, ascribed the latter to the presence of the former. I feel that the presence of supernumerary teeth in cases of cleft palate is a mere incidence.

Schorr [58] spoke of architectonic and vegetative factors which give rise to congenital deformities. He pointed out that any interference with the process of mapping out the palate in definite directions favors vegetative growth, i. e., growth in all directions, and that in these cases any degree of monstrosity may result.

Grünberg [38] mentioned external and internal phenomena that act on the developing fetus. He believed that malformations of the palate were largely due to internal disturbances which were within the developing fetus proper, including among these heredity.

Bumba [44] suggested that syphilis was the chief internal factor involved. Seeman [12] has dismissed this erroneous concept, obtaining negative Wassermann reactions in cases of submucous cleft palate.

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56. Ahlfeld: Die Missbildung des Menschen, Leipzig, F. W. Grunov, 1880.
57. Warnekros: Arch. f. Laryngol. u. Rhinol. **21**:144, 1909.
58. Schorr, George: Virchows Arch. f. path. Anat. **197**:16, 1909.

Charles Mayo [59] expressed his interpretation of anomalies in the following words:

As one observes birth defects or anomalies it is apparent that many of them are normal conditions in some lower form of life; e. g., harelip, cleft palate, fissure defects, anomalies in circulation, multiple ureters, dislocation of organs, clubfoot, postnatal dermoids and defects due to changes in the evolution of the intervertebrate to a vertebrate, which largely affects the nervous system—ancephalus, hydrocephalus, spina bifida. Experimentally, changes in the salts in which the eggs of several of the lower forms of life are developed lead to a certain percentage of anomalies. This is undoubtedly the reason why a high type of fish, like the salmon, probably in an evolutionary state, leaves salt water

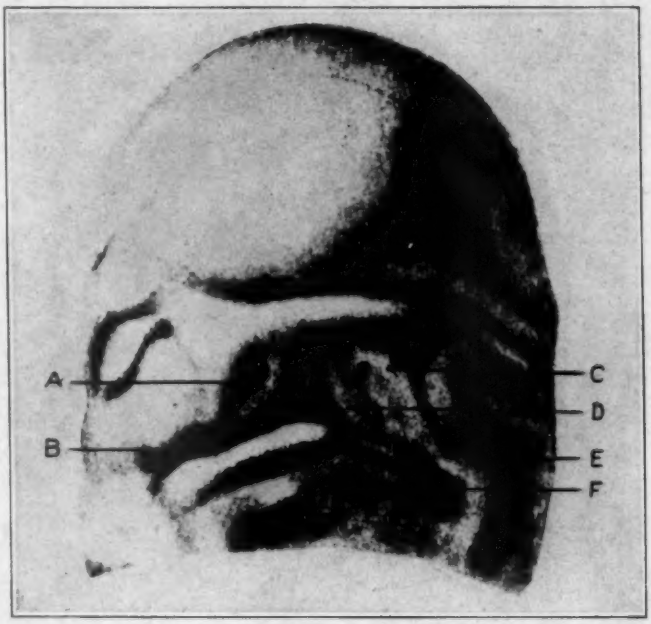

Fig. 1.—Head of a 15 mm. human embryo; × 18. (After Peter.) *A* indicates the anterior naris; *B*, stomodeum; *C*, lateral nasal process; *D*, globular process; *E*, maxillary process; *F*, mandibular arch.

to lay eggs in fresh water. Anomalies in the embryo occur in variation in fluid, especially excessive quantity of hydramnion. It is most probable that the cause of embryonic deformities is due to the changes in the salts of the amniontic fluid in which the egg is developed, just as it happened to be in lower types of life.

As stated previously, despite the fascinating theories advanced in explanation of various anomalies, one is still unable to state definitely their etiology. Each form of anomaly of the mouth or of the palate

59. Mayo, Charles: Discussion of Brophy's paper in Tr. Sect. Surg., General & Abd., A. M. A., 1918, p. 30.

requires a separate embryologic interpretation. The following embryologic interpretation is limited to a consideration of submucous cleft palates alone.

The first branchial arches give rise to the maxillary processes which proceed forward to meet the descending nasofrontal process, the fusion of which forms the upper jaw (fig. 1). From the mesial aspect of

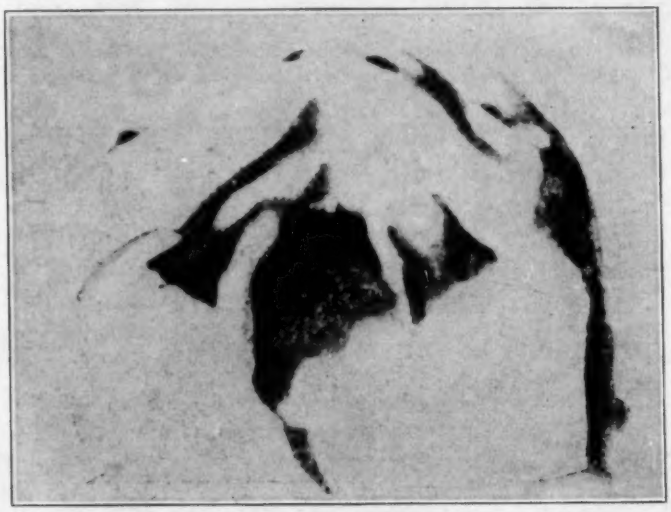

Fig. 2.—Head of a 20 mm. human embryo, from model; × 13. (After Peter.)

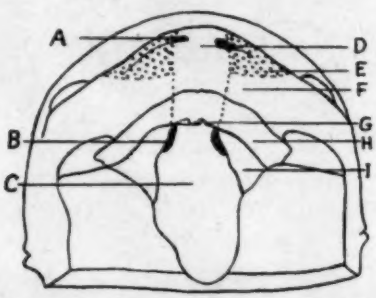

Fig. 2 a.—A diagrammatic sketch of figure 2. *A* indicates the anterior naris; *B*, primitive choana; *C*, roof of the primitive mouth cavity; *D*, middle nasal process; *E*, lateral nasal process; *F*, maxillary process; *G*, papilla palatina; *H*, alveolar process; *I*, palate process.

each maxillary process, ledgelike projections appear and advance toward the midline, where they unite with each other and the septum naris to form the involucrum palati (figs. 2 and 3). An embryonal cleft palate or space lying between the palatal processes exists as a normal condition

in the embryo during the early stages of its development (fig. 4 to be compared with fig. 5). It gradually diminishes in extent as development progresses. These processes begin to unite from before backward in the eighth week of intra-uterine life. At the end of the ninth week, the hard palate becomes fully formed, although the velum remains cleft. A few days later, however, the velum completely closes. This fact, according to Grünberg,[38] explains the rarity of submucous cleft palate and the frequency of clefts involving the posterior part of the palate. Evidently the disturbance of further union of the maxillary elements entering to form the involucrum palati sets in at the same time that union of the maxillary and nasofrontal processes begins anteriorly. This primary union forms the primitive palate which separates the

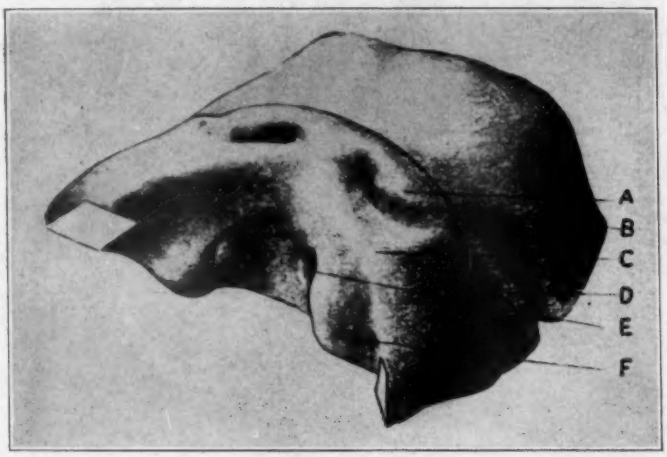

Fig. 3.—The anterior portion of the head of a 15 mm. human embryo, from a model; × 16. (After Peter.) *A* indicates the lateral nasal process; *B*, anterior naris; *C*, maxillary process; *D*, eye; *E*, primitive choana; *F*, palate ridge.

anterior nares from the mouth cavity. That part of the anlage which goes to form the oral mucosa continues its process of union and the soft tissue covering for the palate is thus formed to the tip of the uvula or perhaps to some distance anterior to it. In the meantime, that part of the anlage which enters to form the bony palate and in which the process of union has been inhibited may or may not resume its function while the soft tissue anlage is in an advanced stage of development; in other words, the degree of submucous cleft palate is proportionate to the extent of inhibition of union in the mesodermic elements entering to form the bone. This, according to Peter, explains the varieties of submucous cleft palate.

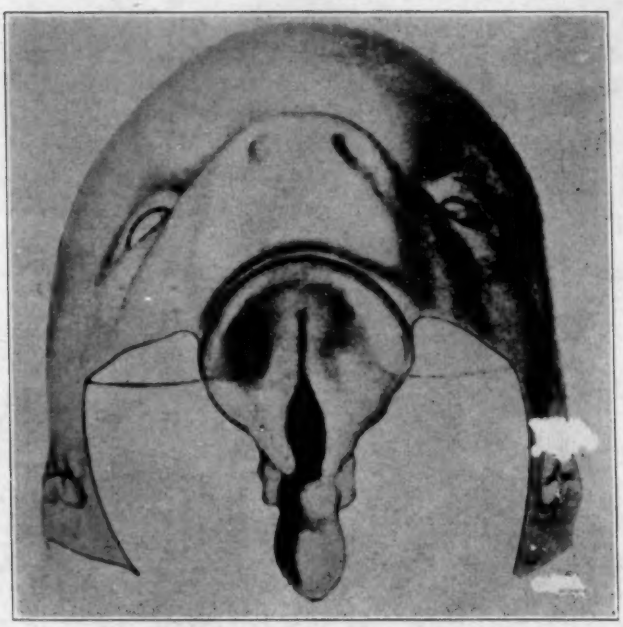

Fig. 4.—Head of 30 mm. human embryo; × 10. (After Peter.)

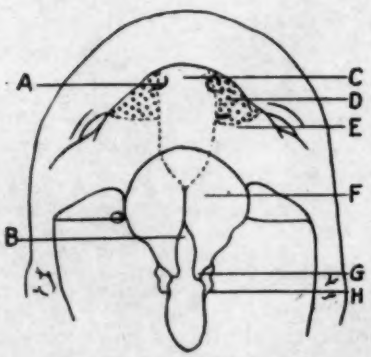

Fig. 4 a.—A diagrammatic sketch of figure 4. *A* indicates the anterior naris; *B*, cleft in the palate; *C*, middle nasal process; *D*, lateral nasal process; *E*, maxillary process; *F*, processus palatinus; *G*, uvula; *H*, palatopharyngeal fold.

APPLIED ANATOMY AND PATHOLOGY

The pharynx is suspended from the base of the skull by the fascia pharyngobasilaris, the attachment of which is shown in figure 6. To this fascia is attached the upper border of the superior constrictor muscle of the pharynx as shown in figures 7 and 8. The pharynx is also held dorsally to the tissue lying on the ventral aspect of the vertebral column by fascial strands extending from the prevertebral fascia. The pharyngeal canal is a muscular tube which moves freely upward and downward while performing its functions. Velopharyngeal closure is accomplished by a sphincter-like mechanism at the lowest part of the nosapharynx, which structure it shuts off from the oropharynx. This

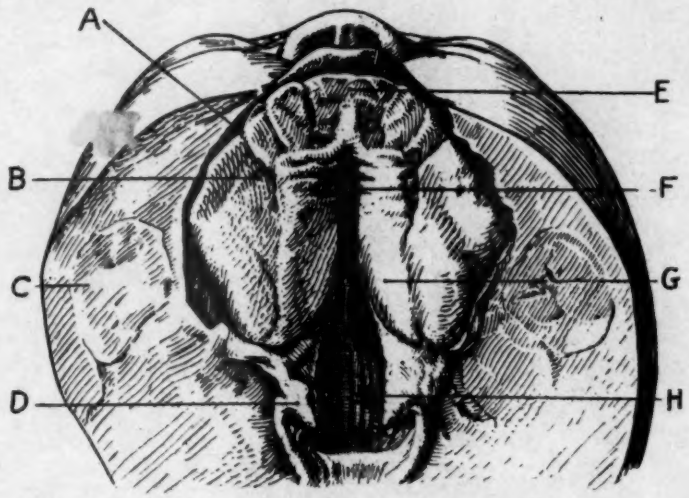

Fig. 5.—Cleft palate in a new-born child from Kollmann's Atlas. (After Peter.) *A* indicates the buccoalveolar sulcus; *B*, processus alveolaris; *C*, cheek; *D*, cleft in uvula; *E*, premaxilla; *F*, septum nasi; *G*, processus palatinus; *H*, base of the cranium.

mechanism is fairly complicated. The structures concerned in causing the closure are the lowermost portions of the nasopharyngeal wall on one side and the velum on the other. The portion of the nasopharyngeal wall which enters into this mechanism is composed of the posterior wall of the nasopharynx; the lateral walls of the pharynx with the eustachian tubes and the salpingopharyngeus bands of the palatopharyngeous muscles. The posterior wall of the nasopharynx where nasopharyngeal closure occurs is formed by the pterygopharyngeal portion of the superior constrictor muscle, which on contraction constitutes what is usually referred to as Passavant's cushion (fig. 7). The velum from the standpoint of

function is divided into two parts, an anterior horizontal portion made up essentially of muscular tissues and a posterior vertical portion which hangs down from the former. The latter segment is formed almost entirely by the uvula. At the junction of these two portions there is an interlacing of the palatal muscles. It is at this point that the levator muscles are attached to the palatine aponeurosis. The levator palati muscle of one side blends with its fellow of the opposite side at their point of insertion into the palatine aponeurosis. So close is their union that the levator palati muscles must be considered as acting as one muscle. The backward and upward elevation of the velum depends

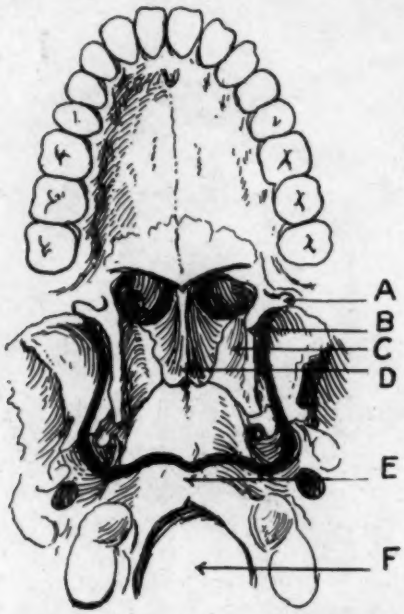

Fig. 6.—The heavy line shows the attachment of the fascia pharyngobasilaris to the base of the skull. *A* indicates the hamular process; *B*, pterygoid plate; *C*, sphenoid bone; *D*, vomer; *E*, occipital bone; *F*, foramen magnum.

exclusively on the position of the insertion of the levator palati muscles. Examination of figure 9 demonstrates this fact. The nasal aspect of this portion of the velum which corresponds with the attachment of the levator palati muscles approximates Passavant's cushion during velopharyngeal closure and forms the anterior segment of this mechanism. At rest, the uvula hangs down as a curtain; in ascending slightly on velopharyngeal closure, it contracts to make the vertical and dependent portion of the velum shorter. This causes the velal segment to become broader, thicker and shorter at the point of insertion of the levators (figs. 9 and 10). The elevation of the pharynx brings these

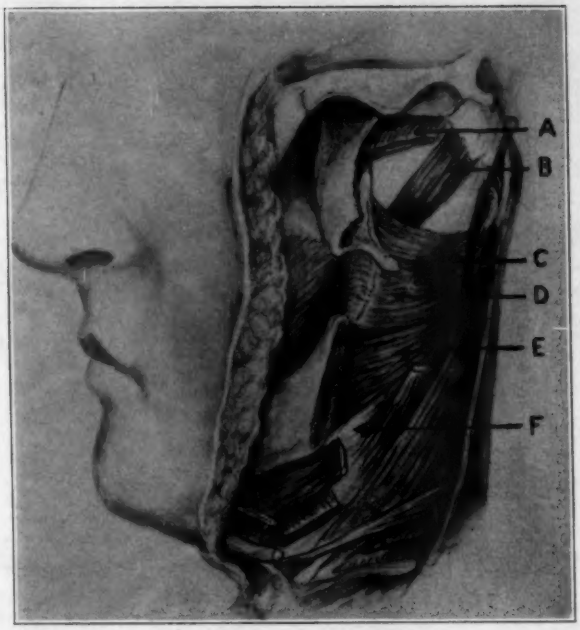

Fig. 7.—Lateral view of the pharynx, showing the superior constrictor muscle. *A* indicates the tensor palati "cut"; *B*, levator palati; *C*, pterygopharyngeus; *D*, buccopharyngeus; *E*, mylopharyngeus; *F*, glossopharyngeus.

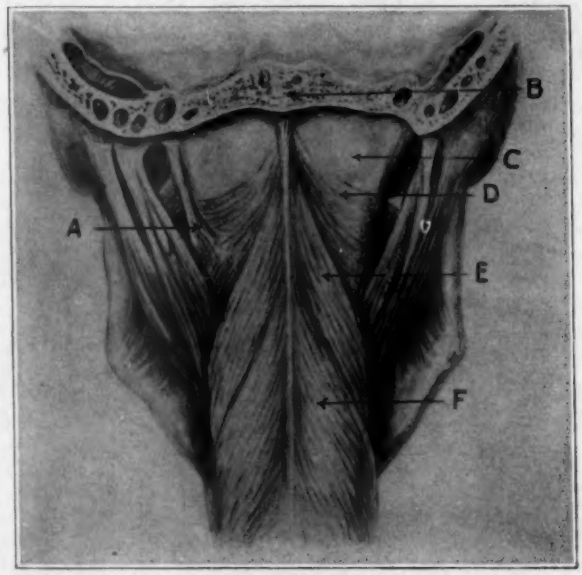

Fig. 8.—View of pharynx from behind. *A* indicates the petropharyngeus muscle; *B*, occipital bone; *C*, fascia pharyngobasilaris; *D*, superior constrictor muscle; *E*, middle constrictor muscle; *F*, inferior constrictor muscle.

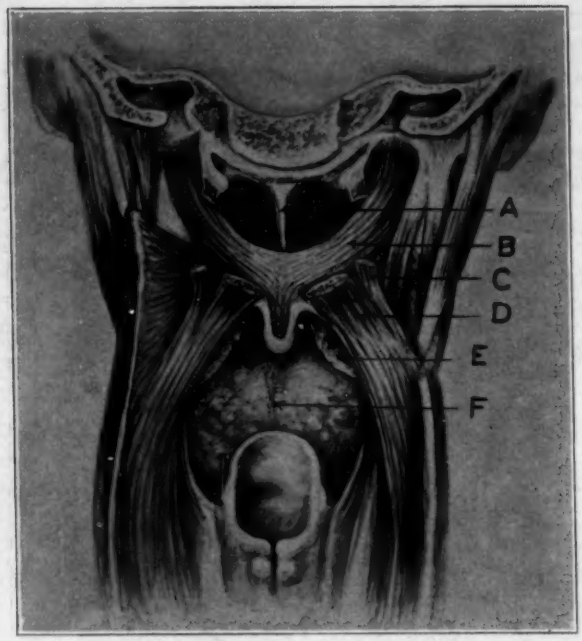

Fig. 9.—View of the palate from behind to show the levator palati muscles. *A* indicates the septum nasi; *B*, levator palati; *C*, salpingopharyngeus "cut"; *D*, palatopharyngeus "cut"; *E*, faucial tonsil; *F*, dorsum linguae.

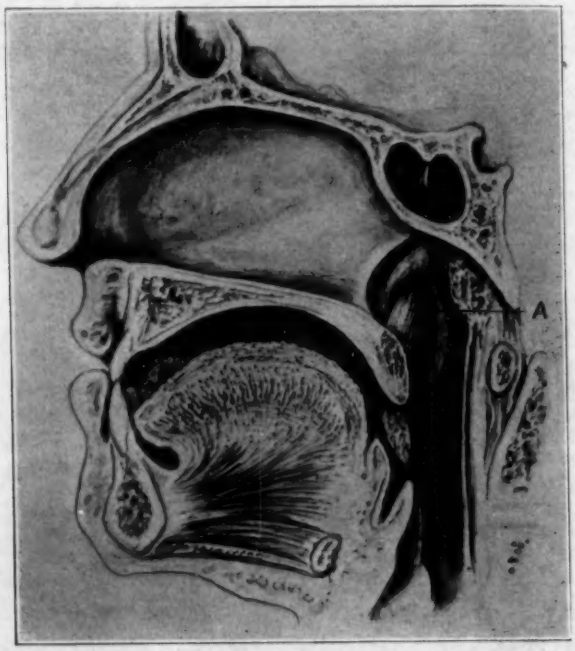

Fig. 10.—Frozen sagittal section with the levator palati muscle dissected. *A* indicates the levator palati muscle.

muscles into proper position, enabling them to close the communication between the oral and the nasal portion of the pharynx. In other words, the superior constrictor muscle of the pharynx pulls the relaxed muscular tissue upward and forward, while the lateral walls approach the midline. The velum is brought upward and backward by the contraction of the levator palati muscles. It is a well known fact that no muscle in the body acts alone without an antagonist to resist its action. It is also recognized that certain muscles in the body are so constructed that one portion of the muscle may exercise a certain function while another portion is moderately contracted or even at rest. The tensor palati muscles raise the velum up to the level of the hamular processes. Beyond this point they act as antagonists to the levators (figs. 11*a* and

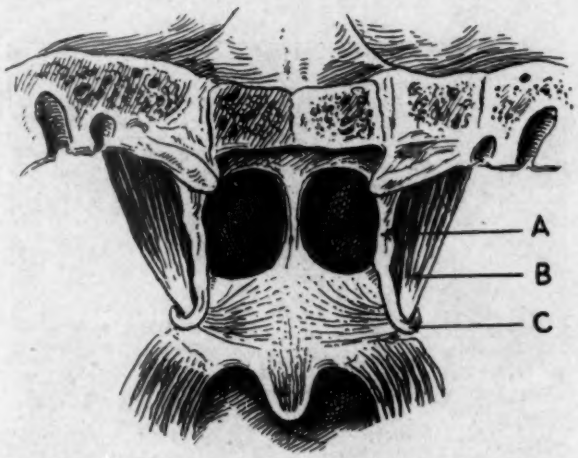

Fig. 11 a.—Sketch showing the tensor palati muscles. *A* indicates the mesial pterygoid plate; *B*, tensor palati muscle; *C*, hamular process.

11*b*). The tensor palati muscles make the palatine aponeurosis tense. Normally, the tensor palati muscles act together. The portion of the tensor palati muscles which arises from the eustachian tubes passes around the hamular process to be inserted into the horizontal plate of the palate bone in the transverse ridges. This portion of the muscle dilates the eustachian tube. The portion which arises from the scaphoid fossa at the base of the mesial pterygoid plate and the spina angularis of the sphenoid bone passes around the hamular process, radiating mesialward in a fan-shaped expansion to be inserted into the palatine aponeurosis and to interlace with its fellow from the opposite side. This portion of the muscle pulls the velum forward and outward, thus making the palatine aponeurosis tense. It is of interest to compare the function of this muscle in a normal palate with one in which a cleft

or submucous cleft is present. In the latter condition, the two tensor palati muscles act separately, thus pulling the velum forward and outward and at the same time turning the tips of the divided uvula inward toward the median line (figs. 12, 13 and 14). Billroth [60] and Agnew [61] recognized this, and advised dividing the hamular process or the tendon of tensor palati muscles when attempting to close palatal defects.

To counteract the forward and outward pull of the tensor palati muscles in cases of cleft or submucous cleft palate, it is necessary to divide the posterior portion of the tendon of the tensor palati muscles. This portion of the muscle constitutes the lateral attachment of the palatine aponeurosis.

The palatopharyngeus muscle is difficult to dissect and more difficult to describe. The muscle has a portion known as the salpingopharyngeus

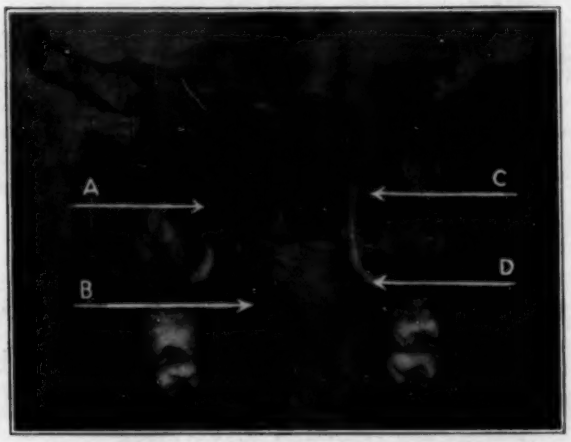

Fig. 11 b.—Photograph of specimen from the Mütter Museum of the College of Physicians of Philadelphia, showing dissection of the velum in man by Dr. Adam Politzer. *A* indicates the levator palati; *B*, palatine aponeurosis; *C*, tensor palati; *D*, hamular process.

muscle which enters to form a muscular band in the lateral wall of the pharynx (figs. 9 and 15). The other portion of this muscle constitutes the palatopharyngeus arch and runs from the velum downward to insert into the lateral wall of the pharynx. The palatopharyngeus muscle, on contracting, depresses the velum, thus acting as a direct antagonist to the levator palati muscles. This muscle, with its fellow of the opposite side, helps form the posterior pillars of the fauces. It is convenient to consider these pillars as a pair of muscles which act

60. Billroth, Theodor: Wien. klin. Wchnschr. **11**:241, 1889.

61. Agnew, D. Hayes: The Principle and Practice of Surgery, Philadelphia, J. B. Lippincott Company, 1889, vol. 2, p. 974.

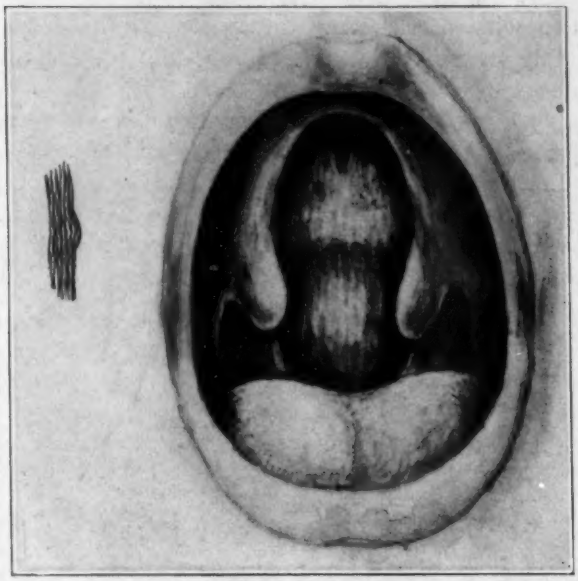

Fig. 12.—View of a case of cleft of the velum showing a well marked Passavant cushion.

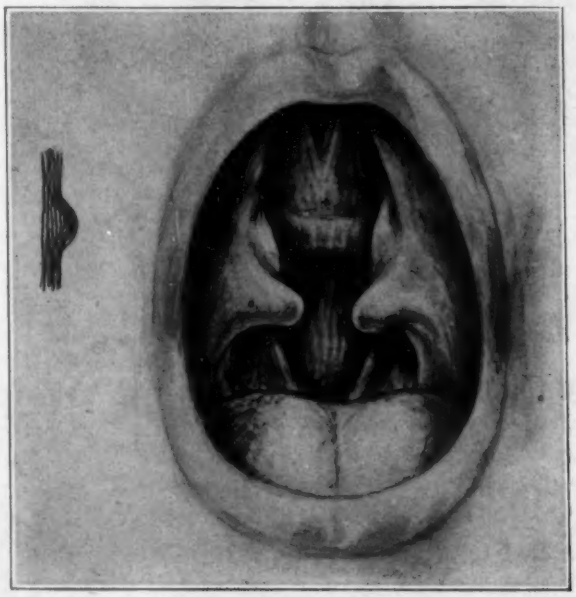

Fig. 13.—View of case shown in figure 12 while the patient attempts to contract the muscles slightly.

synchronously. Their function has more to do with deglutition and vomiting than with speech, although they do draw the velum downward. Further details of their function are omitted since they do not enter into the velopharyngeal closure.

In cases of submucous cleft palate there is a deficiency in the posterior part of the bony palate (figs. 16, 17 and 18). This deficiency may vary from a mere notch to a large V-shaped loss of bone. The palatine mucosa is always intact. The velum in this case is pulled forward with marked shortening in the anteroposterior diameter of the

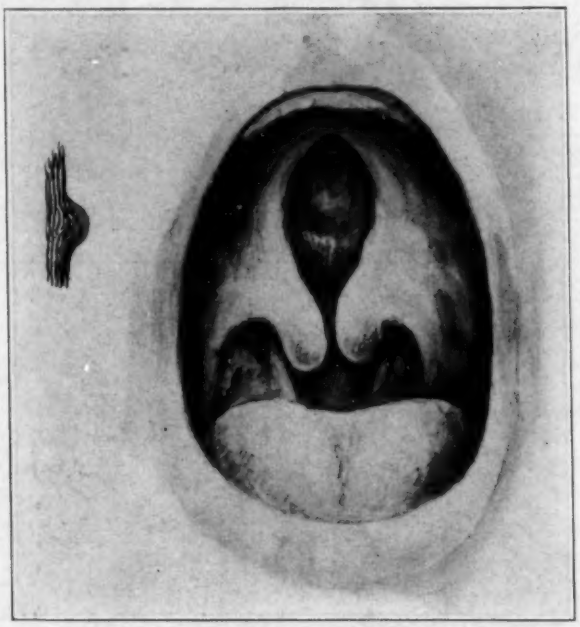

Fig. 14.—View of case shown in figure 13 while the patient attempts complete contraction. Note how the sides of the cleft in the uvula approximate.

palate. The central portion of the velum which covers the deficiency in the bony palate is composed essentially of mucous membrane. This membrane can be seen to move up and down in the roof of the mouth during respiration. The insertion of the levator palati muscles in these cases is displaced forward. Hence they are unable to raise the velum upward and backward to the desired point against Passavant's cushion, at which point, as previously stated, velopharyngeal closure takes place. It must be remembered that there are cases of submucous cleft palate in which there is no loss of bone but a submucous cleft in the muscular tissues and in the palatine aponeurosis which enters to form the velum.

The superior constrictor muscle of the pharynx is composed of the pterygopharyngeus, buccopharyngeus and mylopharyngeus muscles (figs. 7 and 8). This muscle has other accessory muscular slips,[62] among which are the following: petropharyngeus, sphenopharyngeus, petropharyngeus externus, occipitopharyngeus, mastopharyngeus and azygospharyngeus. These accessory muscular strands when present assist the superior constrictor muscle to elevate the pharynx. However, one or more are frequently missing. These muscles take their origin from the parts after which they are named.

The pterygopharyngeus portion arises from the pharyngeal raphe (fig. 7). Starting about 1.25 cm. to 2 cm. below the pharyngeal tubercle

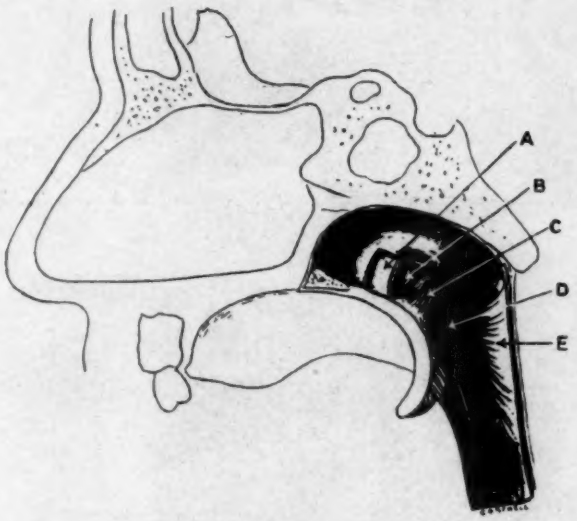

Fig. 15.—Dissection of the nasopharynx in cleft palate. *A* indicates the internal pterygoid plate; *B*, tensor palati; *C*, levator palati; *D*, salpingopharyngeus; *E*, superior constrictor. (From Wardill.)

of the occipital bone, it runs outward and forward to the posterior border of the mesial pterygoid plate and hamular process. This muscle, which is the upper fasciculi of the superior constrictor muscle of the pharynx, forms Passavant's cushion.

The buccopharyngeus, which forms the lateral wall of the pharynx (fig. 7), arises from the pharyngeal raphe and runs outward and upward to the pterygomandibular ligament. Its action produces a slight mesial constriction of the transverse axis of the pharynx.

62. Toldt, Carl: An Atlas of Human Anatomy for Students and Physicians, tr. by M. Eden Paul, New York, The Macmillan Company, 1926.

The mylopharyngeus muscle arises from the pharyngeal raphe. It runs downward, outward and forward to the posterior one fifth of the mylohyoid ridge on the mandible. This muscle has nothing to do with velopharyngeal closure.

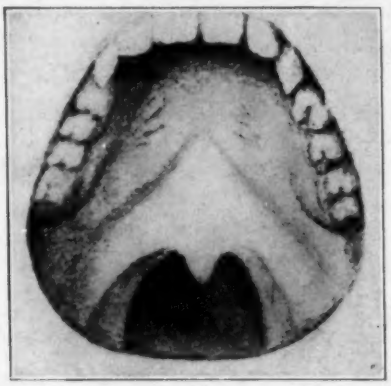

Fig. 16.—Submucous cleft palate. (After A. Brown Kelly.)

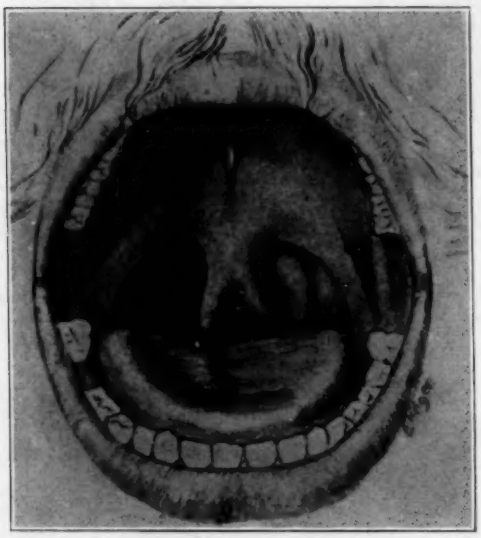

Fig. 17.—Submucous cleft palate with split of the uvula in a man, aged 40. (After Karl Peter.)

The glossopharyngeus portion arises from the pharyngeal raphe, runs obliquely downward, outward and forward and inserts along the sides of the tongue, blended with the transverse linguae muscle.

My conclusions on the function of the pharynx were derived from dissections of human specimens, observations on normal and abnormal palatal cases and experimentations on animals. I believe that the pharyngeal muscles have varied actions, depending on the degree of the elevation of the pharynx. One can readily understand that if the pharyngeal raphe is tense, these muscles will pull in on either side, producing lateral narrowing of the pharynx, since they bring the lateral

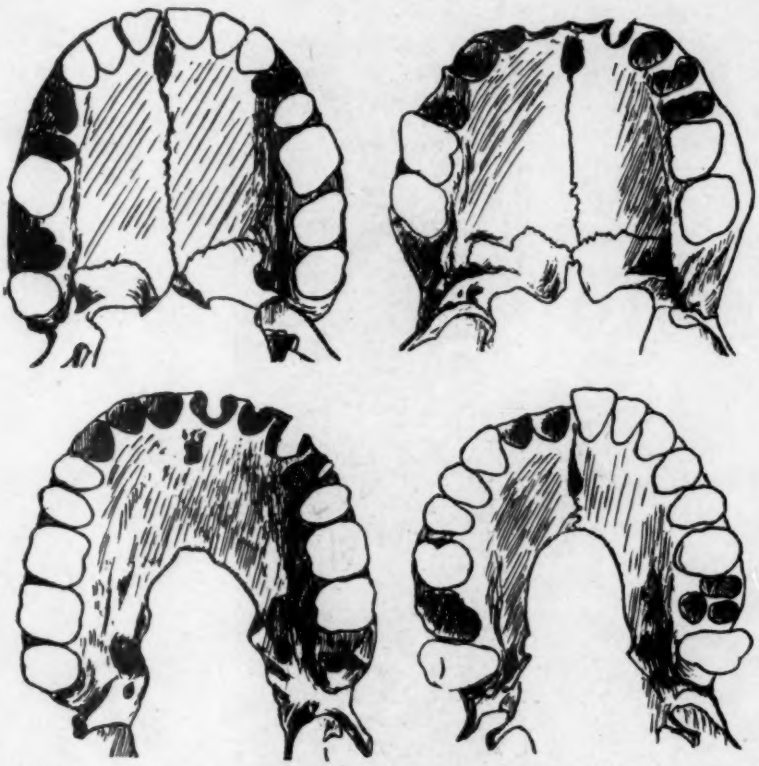

Fig. 18.—Sketches showing the varieties of missing portions from the posterior part of the hard palate. (From A. Brown Kelly.)

walls toward the mesial line. When the entire pharynx is elevated through the action of the suprahyoid muscles the pharyngeal raphe is relaxed. Now contraction of the pterygopharyngeus muscles produces a condition as shown in figure 19. Without the assistance of the elevator muscles of the pharynx, the forward pull of the pharyngeal wall cannot take place, because the weight of the pharynx would prevent the weaker muscles from drawing it forward. As a matter of fact, when the superior constrictor muscle of the pharynx is observed

in a normal case it can be seen to contract by rolling forward and upward in a wavelike fashion, producing a ridgelike bulging.

Until very recently, the walls of the pharynx have not been given sufficient consideration. When one observes velopharyngeal closure through the pharyngoscope or on persons who have suffered the loss of the nose and septum naris, the nasopharynx has a close resemblance to a funnel the apex of which is directed downward. As closure occurs, the walls of the pharynx are pulled forward and inward by the superior

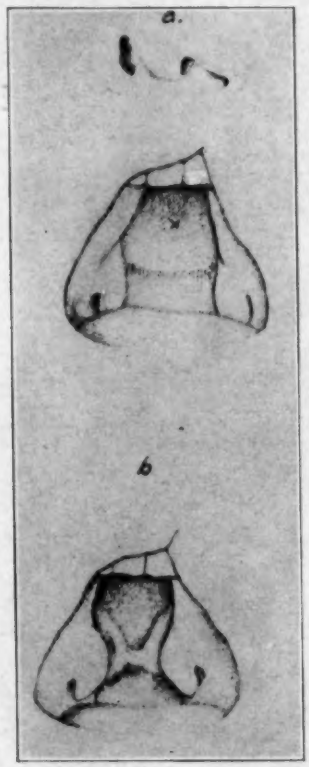

Fig. 19.—In *a*, *X* shows Passavant's cushion at rest in a case of cleft palate. In *b*, the increase in size of the cushion is shown when "ah" is pronounced. (Taken from Passavant.)

constrictor muscles. The longitudinal bands formed by the salpingopharyngeus muscles contract, thus increasing the bulk and thickness of the wall and narrowing the lumen. The anterior segment is drawn upward and backward by the levator palati muscles. In persons with exceptionally well developed velopharyngeal closure the salpingopharyngeus muscle stands out like a band of muscular tissue which extends

from the mesial aspect of the eustachian cartilage, outward on the lateral wall of the pharynx, where it blends with the palatopharyngeus muscle. This muscle has two functions, either of which may be performed independently. That is, it may close the eustachian tube in the one case, or may assist in the elevation of the pharynx in the other. Persons with palates which are normal in length can close the nasopharynx with but slight bulging of Passavant's cushion (fig. 20). In persons whose palates are shorter than normal, velopharyngeal closure can be produced only by more active contraction of that portion of the pharyngeal wall which constitutes Passavant's cushion. It is remarkable how much development may occur in the pterygopharyngeus muscle following successful cleft palate operation or in cases in which the velum is stiff and insufficient. Likewise, the superior constrictor muscle of the pharynx can be forced to undergo marked development by continuous use.

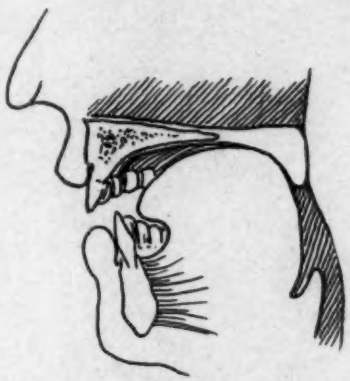

Fig. 20.—Sketch showing how normal velopharyngeal closure occurs. Note the forward movement of the posterior pharyngeal wall manifest by bulging of Passavant's cushion. The vertical portion of the velum is shown to assist in the sphincteric closure.

Velopharyngeal closure is complete in normal persons during the acts of swallowing, gargling and whistling or in the pronunciation of certain letter sounds which are free from nasal intonations. A simple test to determine whether the velopharyngeal closure is complete may be made by holding a cold mirror in front of the anterior nares while the patient forms the letter sound "ah"; if closure is complete the mirror will be unclouded.

The palate has a copious blood supply derived from the nasopalatine vessels, the posterior and accessory palatine vessels and branches from the pharyngeal anastomosis. The nasopalatine arteries emerge from the anterior palatine foramen and anastomose with the posterior palatine

arteries. The descending palatine arteries leave the greater palatine foramina and pursue a tortuous course within the palatine mucoperiosteum, supplying the hard palate, the alveolar processes and the gum tissues. The branches which supply the hard palate anastomose freely with each other, but not with those of the opposite side. The accessory palatine arteries leave the accessory palatine foramina and pass backward to anastomose with the terminal branches of the pharyngeal anastomosis. They supply the velum. The pharyngeal anastomosis is formed by the terminal branches of the lingual, facial and ascending pharyngeal arteries.

The bony palate is said to have an independent blood supply,[63] and is thus protected from necrosis following cleft palate operations. Following ligations of one or both external carotid arteries, collateral circulation is quickly established sufficient to nourish the tissues.

Division of one or both posterior palatine arteries is frequently done in palatal operation. Collateral circulation immediately takes care of the blood supply. It is possible to divide the anterior and posterior palatine arteries in doing the "push-back operation" with perfect safety to the life of the flap, providing that an interval of one week is allowed to elapse before the second stage of the operation is done. There are fewer mucous glands in the palate of children than in adults. This explains the delicacy of the palatine mucoperiosteum in children and its inability to withstand surgical insult.

The nerve supply of the palatoglossus, palatopharyngeus, salpingopharyngeus, azygos uvulae and the superior constrictor pharyngeus muscles is derived from the pharyngeal plexus. This plexus is formed by branches from the glossopharyngeal, spinal accessory and pneumogastric nerves. The tensor palati muscle [64] is supplied by the mandibular branch of the trigeminus. The levator palati [64] is supplied by the bulbar root of the spinal accessory nerve, which contains the inferior rootlets of the pneumogastric nerve.

DIAGNOSIS AND SYMPTOMS

The diagnosis of congenital shortening of the palate can be accurately determined only after the child makes efforts to speak. On examining children before the age of speech, one may observe that the velum is unable to approximate the pharyngeal wall to secure the desired velopharyngeal closure. The final diagnosis cannot be definitely established until a considerable period of time after the child develops articulate speech. Children with congenital shortening of the palate usually learn to speak at a later period than normal children. These children speak

63. Berry, James, and Legg, T. Percy: Harelip and Cleft Palate, Philadelphia, P. Blakiston's Son & Company, 1912, p. 34.

64. Rich, A. R.: Bull. Johns Hopkins Hosp. **31**:307, 1920.

very indistinctly, owing to the inability of the velum to approximate the pharyngeal wall to produce the desired velopharyngeal closure. It is true that as these children develop and make persistent efforts to speak, their speech becomes more and more distinct, depending on their ability to develop a mechanism to compensate for this velopharyngeal insufficiency. They develop the compressor naris muscles and a hypertrophy of the muscles which produce the velopharyngeal closure and they frequently develop a hypertrophy of the faucial and pharyngeal tonsils.

The diagnosis is made from the following symptoms:

1. Rhinolalia aperta or open nasalizing. This begins at the period when the subject began to talk. The vowels take a nasal tone, i. e., the letter "a" becomes "an," etc. The consonants become very much altered, with the exception of the letters "m" and "n" which retain their characteristic sounds. Sigmatismus or the inability to produce the letter sound "s" becomes very pronounced in these cases.

2. These patients suffer from shortness of breath in speaking due to the loss of air through the nose.

3. They are unable to whistle.

4. Hissing becomes impossible.

5. Patients are unable to blow out a candle flame from a distance, unless they have developed a method of compressing the nares.

6. Breathing by mouth becomes a habit, giving these patients a characteristic vacant expression.

7. Movements of the facial muscles, more especially the compressor naris and the corrugator superciliary muscles, become pronounced. These patients are referred to as "face talkers."

8. Hearing may be diminished, owing to improper ventilation of the middle ear. Nasal catarrh is frequently present.

9. Deglutition may become disturbed in children. When these patients become older, they may have difficulty in swallowing.

10. These patients talk very rapidly, and avoid difficult words.

11. Intra-oral examination reveals a distinct velopharyngeal insufficiency. The patient is unable to shut off the nasopharynx from the oropharynx because of the anteroposterior shortening of the palate. Here the shortening may be the result of a shortened hard palate or a shortened velum, or both. The superior constrictor muscle of the pharynx may be weak.

12. Intranasal examination by the pharyngoscope reveals an insufficient velum, which is unable to shut off the nasopharynx.

13. The uvula is bifid in some cases.

14. Palpation may reveal a submucous cleft of the hard palate; this may vary from a mere notch to a considerable V-shaped deficiency in the median portion of the bony palate (fig. 18). The palatine mucosa is found intact. The submucous cleft may involve the velum alone, the hard palate being intact.

15. Irregularity and crowding of the teeth, or congenital absence of the second maxillary incisor tooth, or the association of cleft lip, or the evidence of an intra-uterine healed cleft of the velum may be present.

The palate varies in length with the age, sex, race, build and general characteristics of the individual. Not infrequently, a history of defects in speech or even a case or two of congenital anomalies in the forebears is disclosed on taking a careful family history. If the palate assisted by the superior constrictor muscle of the pharynx is unable to shut off the nasopharynx, it must be considered a short palate. While complete velopharyngeal closure is essential for normal function, this mechanism is not always hermetically sealed during speech, since a certain amount of air escapes through the nostril as certain letter sounds are produced. It is interesting to mention here that Schmidt [65] determined by experiments that speech could remain normal while there was a rubber tube with a lumen of 6 mm. between the velum and the pharyngeal wall, but when a tube of a larger lumen was inserted speech became nasal. This shows that the superior constrictor muscle of the pharynx is capable of overcoming a certain amount of insufficiency which if increased beyond 6 mm. cannot be overcome by this muscle. The shortness of the palate determines the degree of insufficiency. The most reliable test is to hold a piece of cotton in front of the anterior nares while the patient makes efforts to pronounce non-nasal letter sounds. The escaping air makes the cotton move when this mechanism is insufficient. In diagnosing congenital insufficiency of the palate, the condition should not be confused with palsy of the velum, stomatolalia and speech defects, the result of loss of the teeth or in faulty lingual articulation observed in some cases after successful cleft palate operation. In all of these cases, the velopharyngeal closure is normal.

Palsy of the palate is easily recognized by the absence of the palatal reflex, the inability of the velum to move and its lack of response to faradic stimulation.

Stomatolalia or so-called "rhinolalia clausa" is a condition of speech in which the letter sounds lack their necessary nasal resonance, a quality of the speaking voice which determines the supreme elegance of a spoken language.

Defects of speech, the result of the loss of teeth and faulty habits in the use of the tongue, are easily recognized. Loss of the teeth can easily be restored by dentures. Faulty habit in using the tongue can be corrected with methodic speech training.

TREATMENT

In reviewing the literature, one is constantly impressed by the fact that the early pioneers in the field of palatal surgery recognized that perfect speech could be obtained only when the velum was brought in contact with the posterior wall of the pharynx. Many ingenious methods

65. Quoted by Gutzmann (footnote 20).

were devised to accomplish this. One group exerted their efforts to lengthening the palate; another group sought to bring the wall of the pharynx forward, while a third group advised the use of artificial vela. Even today, many surgeons, after failing to obtain a good functional result when the repair was surgically perfect, advise dividing the sutured velum so as to permit the insertion of an artificial velum. The artificial vela of Suersen, or Kingsley, or any modifications of these vela are generally employed. These vela are constructed from soft or hard vulcanite, the latter being the most serviceable. Whenever appliances are instituted for treating insufficiency of the palate, the patient should be informed of the necessity of renewing them from time to time. Through continuous use and due to the fact that the palatal muscles and the superior constrictor muscle of the pharynx hypertrophy, such contrivances lose their relationship with the surrounding structures and new appliances become necessary in order to obtain proper velopharyngeal closure. Obturator-vela may be indicated occasionally in adults, but are rarely necessary in children unless the condition warrants no other method of treatment.

Passavant's [3] observations on speech after successful closure of the cleft palate led him to ascribe nasal intonation in these cases to the inability of the velum to approach the pharyngeal wall. In 1862, he suggested partial union of the velum with the pharyngeal wall to obtain the proper closure. He advised the introduction of sutures on either side of the freshened palatopharyngeus muscles and to a corresponding area on the posterior wall of the pharynx. Passavant also mentioned that union of the palatopharyngeus muscles for a certain extent in the midline may be combined with staphylorrhaphy. However, in 1865, he outlined three different operations to secure the necessary approximation.

The first of these operations consisted in uniting the palatopharyngeus muscles for a certain distance in the midline so as to raise the posterior portion of the velum and so displace it backward to approximate the pharyngeal wall. The upper portions of the mesial border of the posterior pillars were freshened and united by suture after the lateral incisions were made into the velum (fig. 21). This union was obtained in three cases for the distance of 1.25, 2 and 2.5 cm., respectively. Speech was only slightly improved in these cases, nasal intonation persisting.

The second operation which he called staphylopharyngorrhaphy consisted of producing velopharyngeal adhesion. Passavant made a transverse incision across the velum; through this incision, he introduced a spatula bent on the flat. This enabled him to evert the velum permitting its free border to be directed forward. The velum was freshened on its free border for a distance of eight lines in length and five lines in depth;

a corresponding area on the pharyngeal wall was then freshened at the point where velopharyngeal closure was desired. The free border of the velum was then approximated to the posterior wall of the pharynx and sutured (fig. 22). This procedure, as pointed out by Passavant, gave better results than his former method.

Staphylopharyngorrhaphy as suggested by Passavant must be condemned. Through its use stomatolalia is established, normal ventilation of the eustachian tubes is interfered with, and the accumulation of mucous secretions in the nasopharynx is favored.

The third operation consisted of making lateral incisions 1 inch long on either side, mesial to the hamular process, and running anteriorly parallel with the alveolar margin. The anterior extremities of

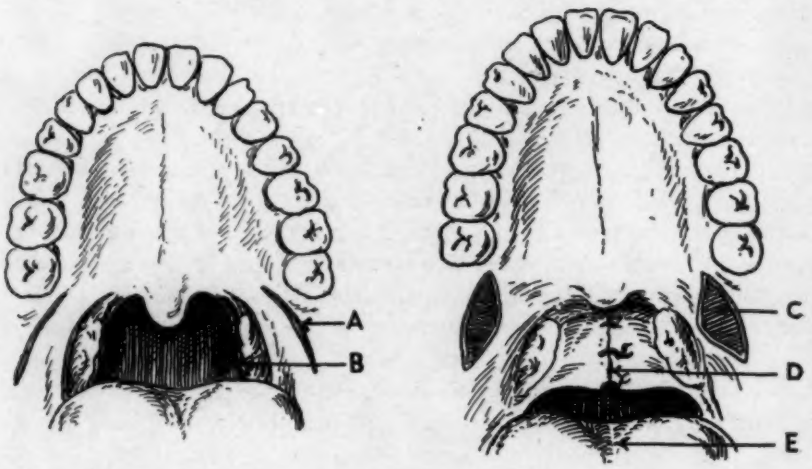

Fig. 21.—Artist's conception of Passavant's operation for suture of the palatopharyngeus muscles. *A* indicates the lateral incision; *B*, freshened border of the posterior pillar; *C*, area to close by granulations; *D*, line of sutures; *E*, dorsum linguae.

these two incisions were joined with a third incision running transversely across the palatine mucoperiosteum. The quadrilateral flap inscribed by these three incisions with its base directed posteriorly, in continuity with the velum, was freed from the underlying bone (fig. 23). The entire velum was displaced backward, and held by suture to the posterior pharyngeal wall. At a subsequent operation, the defect in the hard palate was closed by some autoplastic method. Passavant reported satisfactory results from this procedure.

Nevertheless, in 1878, he condemned all his previous operations and devised one for the correction of velopharyngeal insufficiency. He now proposed a procedure to obtain a bulging of the posterior pha-

ryngeal wall. This was accomplished by inscribing a quadrilateral flap on the posterior wall of the pharynx mesial to and above the level of the eustachian tubes, with the base in connection with the mucous membrane, covering the superior constrictor muscle of the pharynx. This flap was separated from the underlying tissue and folded over on the raw surface, being held in position by sutures (fig. 24). A shelflike projection was thus made on the posterior wall of the pharynx. The sides of this ridge were then united by suture to a corresponding area on the lateral walls of the pharynx. The tissue covering the sphenoid bone which was used to fashion this projection was found to be inadequate for this purpose, as the shelf in time gradually disappeared.

Finally, Passavant made use of an obturator modeled after the shape of a shirt stud (fig. 25); this he inserted into a button-hole incision

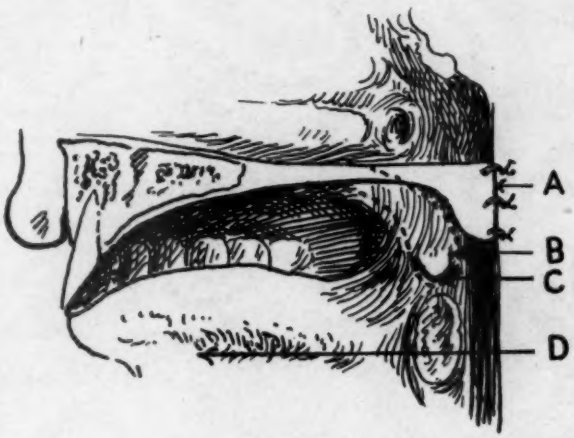

Fig. 22.—Artist's conception of Passavant's operation to produce staphylopharyngorrhaphy. *A* indicates the velum and posterior pharyngeal wall sutured together; *B*, the dotted line to show position of velum; *C*, the heavy line to show free border of velum which has been freshened; *D*, the tongue.

running transversely across the middle of the velum through its full thickness. The purpose of this plan was to secure backward displacement of the soft palate and thereby to establish the desired closure.

In 1864, Simon [14] suggested that the nasal speech which persists after successful cleft palate operations might be corrected by inserting gum balloons into the nostrils or by applying a pince-nez device on the alae nares so as to shut off the escaping air. These methods proved to be inadequate, as the formation of the letter sounds lacked the proper nasal resonance.

In 1867, Simon suggested the use of Dieffenbach's osteal uranoplasty to obviate velopharyngeal insufficiency. His operation consisted of

freshening the edges of the cleft in the hard and soft palate, along with scarifying the edges of the bone. Lateral incisions were made on either side into the hard palate down to the bone at a distance varying from one and one-half to two lines from the alveolar margin. The mucoperiosteum surrounding these lateral incisions was then elevated from the underlying bone. The bony palate was sawed through at the site of the lateral incisions (fig. 26), beginning behind at a point about one-half line mesial to the hamular process, thus dividing the palatal processes from their alveolar connection. The division through the bone was extended anteriorly as far forward as the termination of the lateral incisions. A lever was then introduced into these lateral incisions in the bone and the anterior attachment of the palatal processes were fractured to produce their mesial displacement. This supplied two

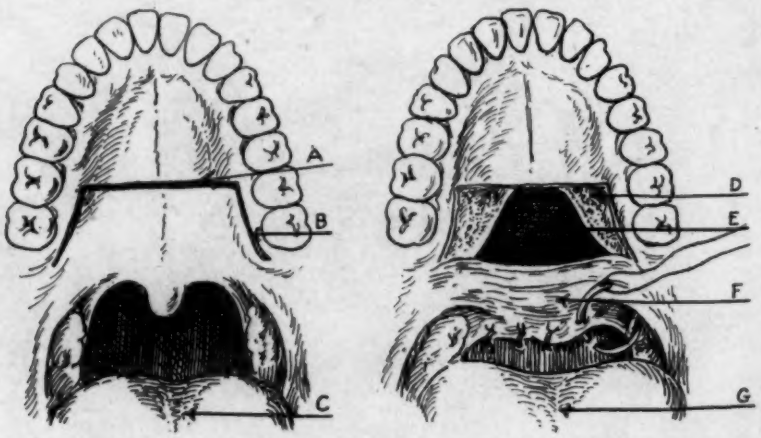

Fig. 23.—Artist's conception of Passavant's operation for backward displacement of the velum. *A* indicates the transverse incision; *B*, lateral incision; *C*, dorsum linguae; *D*, denuded bone; *E*, nasal cavity; *F*, displaced velum; *G*, dorsum linguae.

freely moving osteomucous flaps which were attached anteriorly to the palatine mucoperiosteum situated behind the incisor teeth, and posteriorly with the soft tissue in continuity with the velum. These flaps were then approximated toward the median line and united with sutures applied through the mucoperiosteum.

Osteal uranoplasty was first suggested in 1826, by Dieffenbach,[66] and later by Wutzer [67] and Bühring [68] in 1850. In 1873, Fergusson [69]

66. Dieffenbach: Litt. Ann. d. ges. Heilk. **4**:145, 1826; Die operative Chirurgie, Leipzig, F. A. Bockhaus, 1845, vol. 1, p. 856.

67. Wutzer, C. W.: Deutsche Klin. **2**:26, 1850.

68. Bühring, Julius: J. d. Chir. u. Augenh. **9**:325, 1850; Deutsche Klin. **2**:472, 1850.

69. Fergusson, Sir William: Lancet **2**:784, 1873; **1**:298 and 883, 1874.

modified this method. The operation, although formidable, was performed from time to time by different cleft palate surgeons on the European continent and in America, among whom Mears and Roe of Philadelphia are worthy of mention. Brown of Milwaukee and Davis [49] of Philadelphia have recently popularized osteal uranoplasty, using it as their method of choice in suitable cases of cleft palate.

Paul,[70] in 1866, advised broadening the stiffened and narrowed velum resulting from staphylorrhapy by making repeated lateral incisions in the velum. These heal by granulations. This method is based on the same principle of healing by granulations brought out by

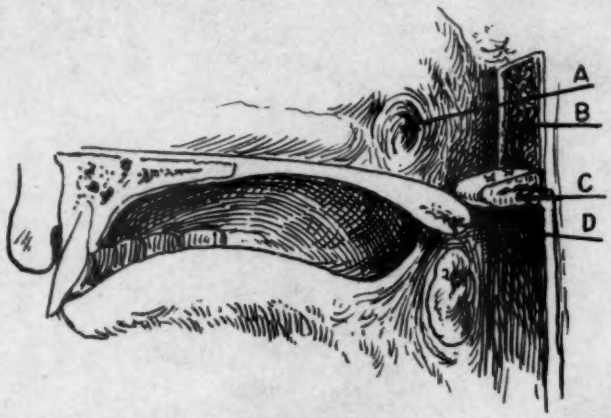

Fig. 24.—Artist's conception of Passavant's operation for making a shelflike projection on the posterior pharyngeal wall. *A* indicates the eustachian tube; *B*, denuded area; *C*, projection; *D*, velum.

Fig. 25.—Passavant's stud-shaped obturator. (After Küster.)

Mettauer [71] of Virginia for performing staphylorrhaphy (figs. 27 and 28). The rationale of this method was that the granulations resultant from the healing of these wounds added bulk to the velum, producing an increase in the tissue and thus assisting velopharyngeal closure.

Mason,[72] in 1869, advised for correction of nasal speech after cleft palate operations otherwise successful the making of lateral incisions

70. Paul, Hermann Julius: Arch. f. klin. Chir. **7**:199, 1866.

71. Mettauer, John B.: Am. J. M. Sc. **21**:331, 1837.

72. Mason, Francis: Lancet **2**:198, 1869; **2**:578, 1874; St. Thomas Hosp. Rep. **2**:271, 1871; On Cleft Palate and Harelip, London, J. & A. Churchill, 1877, p. 114; The Retrospect of Practical Medicine and Surgery, 1870, p. 123.

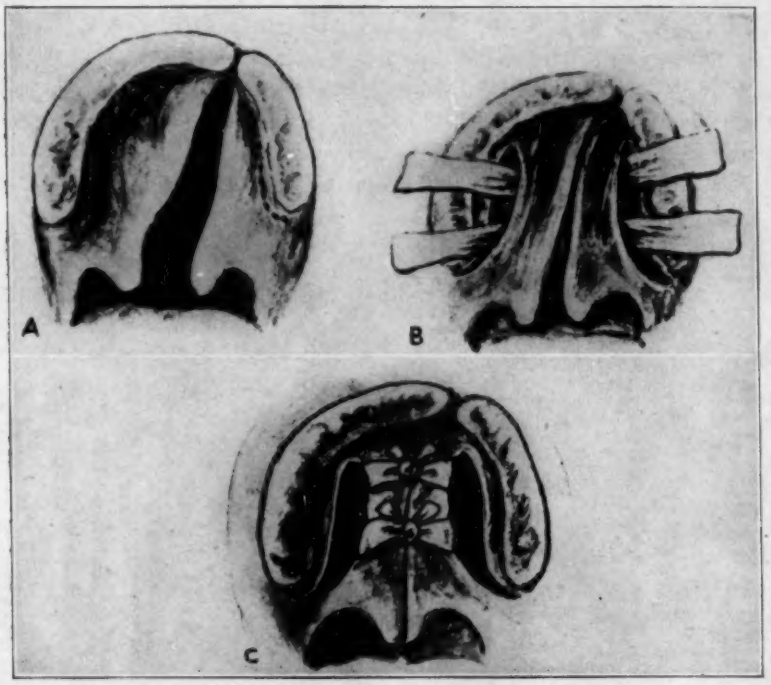

Fig. 26.—Steps in osteal uranoplasty.[7] (After Davis.) *A* is a semidiagrammatic sketch showing unilateral cleft palate and the outline of incisions (slight extension of the Langenbeck incision) used in loosening the mucoperiosteal flap on the side attached to the vomer and loosening the flap containing the rudimentary horizontal process of the maxilla and of palate bone on the opposite side. The latter incision is carried through the periosteum after which the horizontal processes are cut through with a very thin chisel. *B* is a semidiagrammatic sketch showing a mucoperiosteal flap loosened on the patient's right side and a flap containing bone on the patient's left side. In the two-stage operation, tapes are passed around both flaps and tied so as to hold the medial margins of the flaps in apposition, or nearly so, care being taken not to exert sufficient pressure by the tapes to shut off circulation. Iodoform gauze packs are used in the lateral incisions for twenty-four hours. *C* is a semidiagrammatic sketch showing the tapes tied so as to hold the medial margins of the flaps in apposition. Iodoform gauze packs are used in the lateral incisions for twenty-four hours. One tape is removed on the third or fourth day, the remaining tape on the fifth or sixth day. On the seventh day, the mucous membrane is removed from the margins of the clefts and sutures applied.

into the full thickness of the velum internal to the hamular process, to run posteriorly through the free border of the velum. The result of such a procedure altered the stiffened velum into a large floating uvula which was unable to approximate the pharyngeal wall. Mason gives the following description of his operation:

A small curved spatula is first placed behind the soft palate; it keeps the part steady and also serves as a point d'appui. A sharp pointed knife is then introduced from before backward (*A*, fig. 29) in about the position of the inner edge

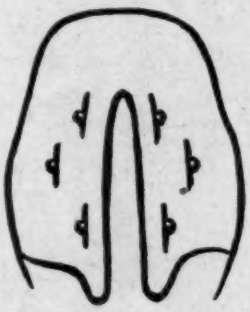

Fig. 27.—Mettauer's incisions to produce healing by granulations in clefts of the velum. (From Mason.)

Fig. 28.—Diagrammatic sketch to show the velum after the application of sutures. The shaded areas to close by granulation tissue. (Modified from Mason.)

of the hamular process in the normal palate (*D*), and the soft palate is cut completely through from above downwards from *A* to *B*. The same is done on the opposite side, and the operation is then concluded. In the first few cases on which I operated, I hemmed the mucous membrane back and front as indicated in the diagram *E*, but I have long since abandoned this practice, as unnecessary for when the parts unite, they do so at the V-shaped angle where these are in immediate contact (dotted line *F*). The operation is quite simple and may be repeated as often as necessary; is perfectly free from danger, and almost painless. The rationale of the proceeding is easily explained. The palate becomes converted into a huge uvula, so to speak. It is shortened and puckered up, the

point *B* being drawn to *C* so that if it does not actually touch the back of the pharynx, it approaches so nearly as to divert the current of air to a considerable extent from the nose into the mouth and thus greatly obviates the disagreeable guttural voice that is more or less observable in all patients who are subjects of this distressing deformity.

It is evident from Mason's description that his operation gave freedom to the palate because of the complete relaxation obtained by the division of the tensor palati muscles. The incisions used by him undoubtedly divided the opponents to the levator palati muscles and thereby enabled the velum to be more easily raised.

Whitehead [73] of New York, in 1871, attempted to lengthen the velum by adding to it two lateral flaps dissected from the posterior pillars of

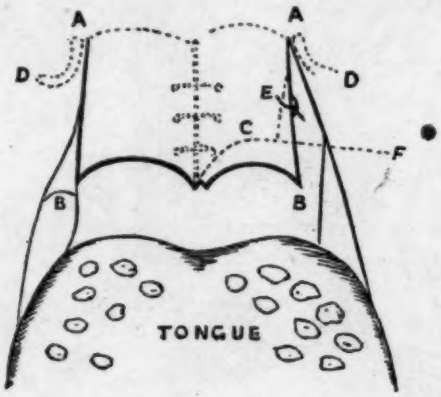

Fig. 29.—Mason's operation for the improvement of speech after cleft palate operations. (From Mason.)

fauces and the lateral walls of the pharynx (fig. 30). He described his procedure in the following words:

About the first week of February, assisted by Dr. Henry Schiff of this city, I endeavored by a difficult and laborious dissection of the palatopharyngeus muscles, to form flaps with which to lengthen the velum palati. Having seized, with a pair of forceps, the palatopharyngeus on the right side very low down, I divided this muscle and a part of the mucous membrane of the prevertebral region, and dissected upward with a pair of curved scissors a flap more than sufficient to form, with a corresponding one on the opposite side, a long and dependent curtain to the new velum.

Makuen, of Philadelphia, has been mentioned by Brophy [74] (figs. 31 and 32) and others as having developed the method by which two

73. Whitehead, William R.: Am. J. M. Sc. **62**:114, 1871.

74. Brophy, Truman W.: Oral Surgery: A Treatise on Disease, Injuries and Malformations of the Mouth and Associated Parts, Philadelphia, P. Blakiston's Son & Company, 1915, p. 709; Cleft Lip and Cleft Palate, Philadelphia, P. Blakiston's Son & Company, 1923, p. 252.

flaps are raised from the palatopharyngeus arch and sutured to the free freshened border of the velum. The works of Makuen [75] do not present this operation. The only remark he made on this subject was in 1901 when he mentioned separating the adhesions between the pillars of the fauces and remnants of the tonsils on either side to overcome velopharyngeal insufficiency. This use of the posterior pillars of the fauces proved to be of no value. It is interesting to mention here that in 1912, Helbing [76] used the posterior pillars to increase the size of the velum as is shown in figure 33. Helbing freshened the mesial border of the palatopharyngeus arch extending this denudation over the intervening portion of the free border of the velum. An incision was then made on either side dividing the posterior pillars as shown at *X*. Side incisions were also made within the velum to release all tension. The freshened edges of the palatopharyngeus arch were united in the midline by suture.

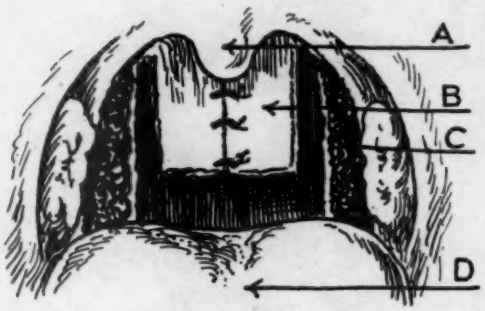

Fig. 30.—Artist's conception of Whitehead's operation, showing the use of the palatopharyngeus muscles in lengthening the velum. *A* indicates the uvula; *B*, posterior pillars sutured in midline; *C*, area to close by granulations; *D*, tongue.

Interference with the palatopharyngeus muscles changes the flexibility of these muscles so that they become more or less rigid, nonelastic bands of tissue which hold the velum downward and prevent it from being elevated beyond a certain point. The use of the posterior pillars in palatal operations is of no value in raising the velum and is a decided hindrance to its free movement during speech and deglutition.

As already pointed out in this paper in the section dealing with anatomy, the uvula only slightly assists in closing the nasopharynx. Velopharyngeal closure is produced by the combined action of the levator palati muscles and the superior constrictor muscle of the pharynx. The

75. Makuen, G. Hudson: Am. Med. **2**:532, 1901.

76. Helbing, Carl: Berl. klin. Wchnschr. **49**:980, 1912; Allg. med. Centr. Ztg. **81**:21, 1912; Die Technik der Uranoplastik, im Payr und Kuettner: Ergebnisse der Chirurgie und Orthopedie, Berlin, Julius Springer, 1913, vol. 5, p. 81.

Fig. 31.—Manner of lengthening palate by utilizing portion of pharyngeal muscles. The posterior view is shown with wires in place. (From Brophy.)

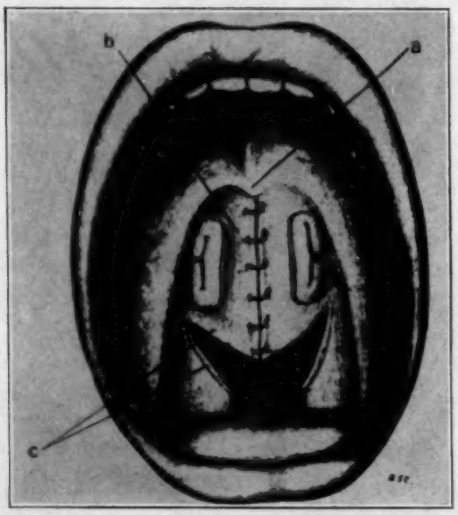

Fig. 32.—Manner of lengthening the palate by utilizing the palatopharyngeal muscles, after Makuen's method. *a* indicates the end of the uvula muscle; *b*, right palatopharyngeal muscle; *c*, incision made through two-thirds the width of the muscle. The lead plates are shown with the silver wires twisted so as to approximate the edges of the wound. (From Brophy.)

formation of a long uvula merely adds a dependent pendulum from the free border of the velum and in some cases may be of more annoyance to the patient than of help.

Schoenborn,[77] impressed by the experiments of Trendelenberg, in 1875 performed velopharyngoplasty to obtain the desired function of the nasopharynx during speech. He outlined a rectangular flap from 4 to 5 cm. long by 2 cm. wide on the posterior wall of the pharynx. This flap was then elevated with the underlying tissue, turned downward on its pedicle, and trimmed triangularly to fit into the cleft of the velum (fig. 34). The velum was freshened and united on either side with the edges of the pharyngeal flap. Schoenborn later decided that the tissue above the superior constrictor muscle of the pharynx was not suitable for his purpose, and in 1886 he took the flap with the pedicle directed upward from the tissue of the superior constrictor muscle. This method was recently revived by Rosenthal,[78] who in 1924 performed velopharyngoplasty.

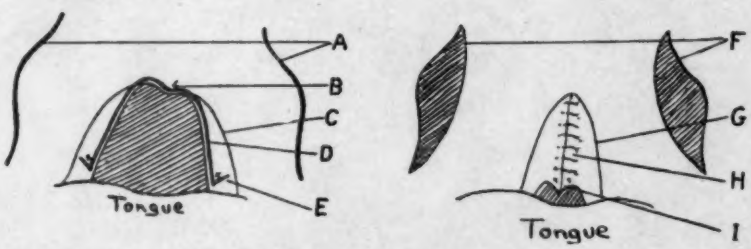

Fig. 33.—Use of the palatopharyngeal arch for lengthening the velum. (After Helbing.) *A* indicates the lateral incisions; *B*, "rest" of uvula; *C*, anterior pillar; *D*, posterior pillars and uvula freshened; *E*, transverse incision into palatopharyngeus muscle; *F*, areas to fill with granulations; *G*, anterior pillars; *H*, newly formed velum; *I*, newly formed uvula.

In 1876, Rutenberg [79] proposed that the pharyngeal wall be brought forward so as to enable it to be reached by the velum, thus producing velopharyngeal closure. In order to secure this, he suggested scarifying a semicircular area measuring from 0.5 to 1 cm. in width on the mucosa covering the pharyngeal wall, and extending from one hamular process to the other. Rutenberg later added that this method was inadequate in many cases, and suggested that in addition to the scarification as previously outlined, the denuded area might be sutured in a longitudinal direction after the method used in colporrhaphy (fig. 35). Scarification may be done either with a knife, the galvanic cautery or chemicals.

77. Schoenborn: Verhandl. d. deutsch. Gesellsch. f. Chir. **4**:235, 1876.
78. Rosenthal: Zentralbl. f. Chir. **51**:1621, 1924.
79. Rutenberg, D. C.: Wien. med. Wchnschr. **26**:815, 839 and 862, 1876.

Whenever the tissues forming the pharyngeal wall are utilized to correct palatal insufficiency, scar tissue is always formed, and this will interfere with the forward pull of the superior constrictor muscle of the pharynx. It has been definitely shown that by systematic speech training the uppermost portion of the pterygopharyngeus muscle can be developed into a much more prominent ridge than can be obtained by any operative procedure.

Forbes,[80] of Philadelphia, in 1879, while closing clefts of the uvula, attempted to lengthen the velum by using a curved incision similar to that used by Nélaton for partial cleft lip operations (fig. 36).

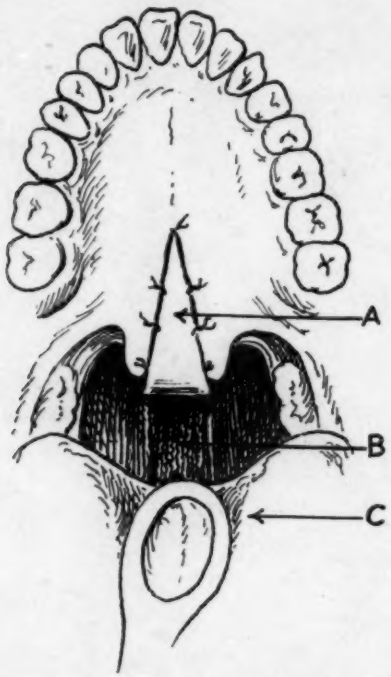

Fig. 34.—Artist's conception of Schoenborn's velopharyngoplasty. *A* indicates the pharyngeal flap trimmed triangularly; *B*, denuded area on pharyngeal wall; *C*, dorsum linguae.

Küster[81] performed an operation in 1882 in which he employed lateral incisions similar to those used by Mirault for cleft lip operations (fig. 37). This procedure, as can be seen from the illustrations, makes a long trilobate uvula. He stated that if this did not produce the desired function sought, the shirt stud-shaped obturator of Passavant might be employed with complete satisfaction.

80. Forbes, W. S.: Tr. Coll. Phys. Philadelphia **1**:70, 1875.
81. Küster, E.: Berl. klin. Wchnschr. **19**:172, 1882.

Fränkel,[17] in 1882, first suggested advancing the attachment of the superior constrictor muscle of the pharynx by altering the position of its insertion. He did not outline any method to secure this, although he did refer to using the same principle employed in treating strabismus.

Wolff,[16] of Berlin, in 1885, advised the use of Schlitsky's soft rubber pharyngeal obturator (fig. 38) after successful cleft palate operations

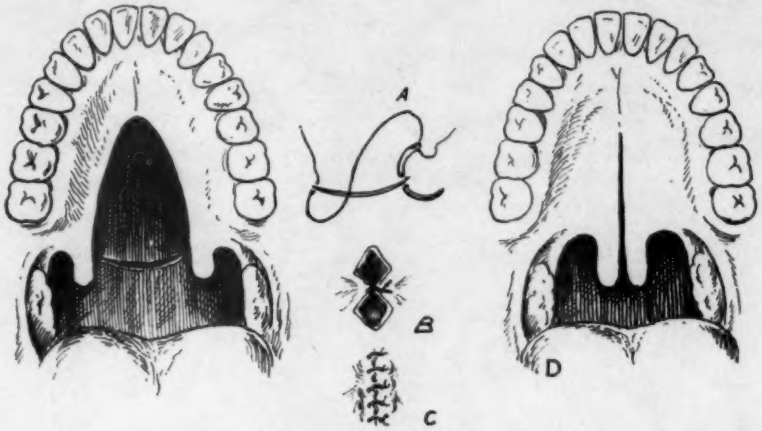

Fig. 35.—Artist's conception of Rutenberg's operation to bring the pharyngeal wall forward. *A* indicates the suture through the extremities of incision to produce vertical approximation; *B, A,* after knotting of suture; *C,* incision sutured vertically; *D* shows how sides of the cleft approximate after vertical suture of incision as shown in *C*.

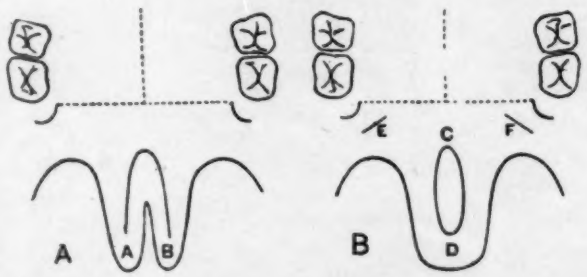

Fig. 36.—Forbes' operation. A diagram of the bifid uvula is shown in *A*. The incision is represented by the line A B. The bifid uvula after operation is shown in *B*. C D indicates the oval wound left to heal by granulation; E F, the incisions for division of the palatine muscles. (From Forbes.)

in all cases in which the velum was too short to reach the wall of the pharynx. He discarded this obturator, however, in 1894 for Hahn's hollow, hard rubber pharyngeal obturator, which except for material was constructed very much as was Schlitsky's obturator.

Billroth,[60] in 1889, was the first to advise dividing the hamular process in cleft palate operations for the purpose of releasing tension on the line of suture. He stated that this permitted the velum to hang freely during its attempts at closure of the nasopharynx.

In 1893, Mears,[9] of Philadelphia, divided the tensor palati, palatopharyngeus and palatoglossus muscles in a case in which the velum was too short to approach the pharyngeal wall. Mears reported that there was marked improvement in the speech of his patient, a young lady who suffered from congenital velopharyngeal insufficiency.

In 1895, Smith,[82] of Nashua, New Hampshire, devised an operation by which uranostaphyloplasty was accomplished with backward dis-

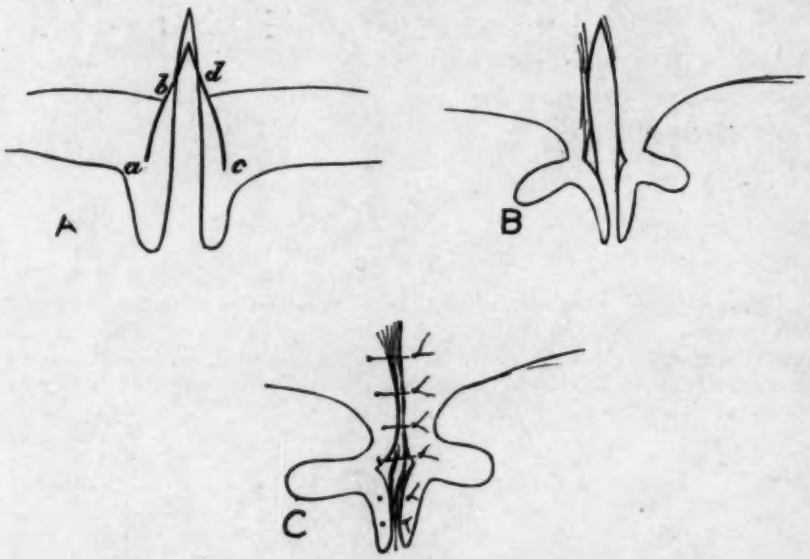

Fig. 37.—Küster's operation. In *A*, *ab* and *cd* are cross-incisions running into the full thickness of the velum. In *B*, the velum is displaced backward and made longer. *C* shows *B* after the application of sutures. (From Küster.)

placement of the velum, permitting it to reach the pharyngeal wall. Smith described his operation in the following words:

> From the anterior end of the cleft an incision is made running outwards and a little backwards towards the alveolus, about one-third the distance to the teeth, when the direction of the incision is changed so that it is carried in a straight line nearly to the posterior extremity of the alveolar process (fig. 39 *A*). The point at which this incision ends varies with the case, care being taken that sufficient tissue is left for the blood supply of the flap. A corresponding incision having been made upon the opposite side, the two flaps thus outlined are dissected

82. Smith, Herbert L.: Boston M. & S. J. **132**:478, 1895; Tr. New Hampshire M. Soc., 1905, p. 227; Am. J. Surg. **20**:65, 1906.

up from the bone, the periosteum being included, and are freed completely from the posterior edge of the palate bones. This last act is very important as only thus can the velum be carried backward and so lengthened. An incision is now made on either side beginning at the point near the last molar where the preceding incision terminated and running along in a curve close to the roots of the teeth as far forward as is necessary or as is deemed safe, sufficient space being left in front for the nourishment of the flaps. This cut usually terminates near the lateral incisors (fig. 39 *B*). There are thus formed two triangular flaps composed of mucous membrane and periosteum which are carried into the median line and

Fig. 38.—Schlitsky's obturator. (After Partsch.)

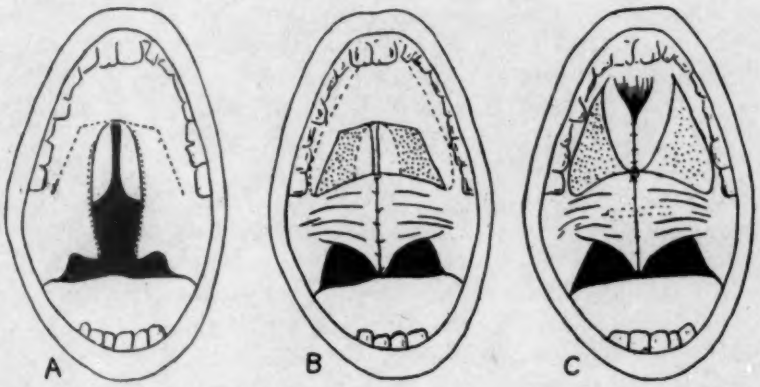

Fig. 39.—Smith's operation for lengthening the velum. (From Smith.)

sutured together. The freshened edges of the velum are now sutured and the posterior edges of the anterior flaps are sewn to the middle of the newly formed velum (fig. 39 *C*). If deemed best, tension can still further be guarded against by extra sutures placed in the velum at the same distance from the edge. In case there seems danger that the palate may be carried too far forward during the process of repair through contraction of the flaps, it has seemed to the writer quite feasible to pass a suture through the periosteum at the posterior edge of the palate bones, and thus anchor the velum at the desired point.

Gutzmann [25] of Berlin, in 1899, advised massage of the velum with an apparatus which conformed with the contour of the palate (fig. 40).

The loop of this instrument was covered with gutta-percha. Gutzmann also advised that systematic speech training should be instituted as an adjunct to any form of treatment for cleft palate.

Gersuny, in 1900, suggested the possibility of advancing the posterior wall of the pharynx to secure velopharyngeal closure by injecting soft paraffin into the retropharyngeal space (fig. 41). This method was modified in 1902 by Eckstein,[83] who substituted hard paraffin. The ill effects arising from the use of paraffin in the retropharyngeal space were later pointed out by Lexer, Warnekros, Röse and von Gaza.

Philip,[30] in 1904, reported favorable results by the use of the faradic current on the posterior pillars. He advised that the electricity be used in conjunction with speech exercises, and suggested this treatment in all cases of congenital insufficiency of the palate.

Fig. 40.—Gutzmann's hand obturator. (After Nadoleczny.) *A* indicates the velum against the posterior wall of the pharynx; *B*, the hand obturator in position.

In 1907, Botey [10] suggested narrowing of the velopharyngeal space by reducing the length of the curve inscribed by the pharyngeal wall at the level where contact of the velum with the wall of the pharynx is desired. To accomplish this, he outlined with silver nitrate on the median part of the pharyngeal wall, an ellipse running with its long axis in the vertical plane (fig. 42 *A*). This ellipse measured from 30 to 40 mm. in its longitudinal diameter and from 15 to 16 mm. in its transverse diameter. The elliptical area was then excised and the edges of the wound united by suture (fig. 42 *B*).

In 1911, Blair,[85] of St. Louis, developed an autoplastic method in which he made use of the side incisions employed by Küster to overcome further shortening of the velum in cases in which the soft palate

83. Eckstein: Berl. klin. Wchnschr. **39**:315, 1902.

was too far forward from the pharyngeal wall. Blair described his operation in the following words:

Küster proposed the plan by means of the incisions *b-b* (fig. 43*A*) of lengthening the velum, but there is rarely sufficient palate tissue to permit this being done without leaving such wide gaps at the site of the lateral incision that subsequent scar contraction renders the velum too tense to move freely. The figure 43*A* shows a modification which obviates the latter difficulty.

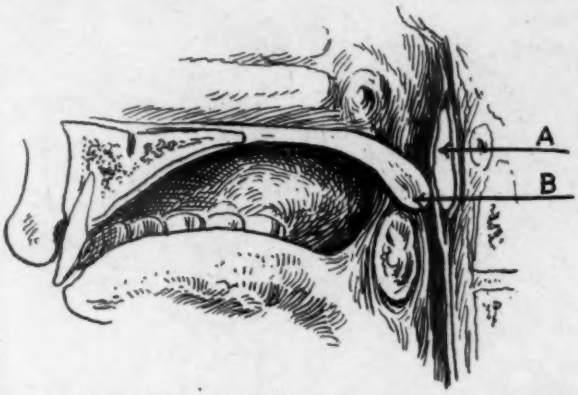

Fig. 41.—Artist's conception to show how the injection of paraffin by Gersuny and Eckstein may produce bulging of the posterior pharyngeal wall. *A* indicates the paraffin in the retropharyngeal space; *B*, the velum.

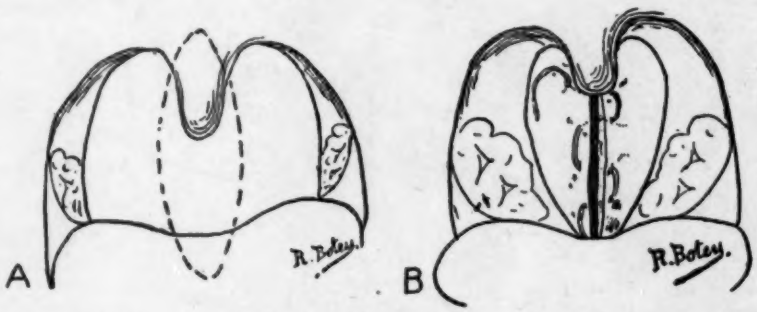

Fig. 42.—Narrowing of the pharyngeal canal. (After Botey.) In *A* Botey shows the elliptical incision made use of in his operation; in *B*, suture of the wound in the posterior wall of the pharynx by the continuous suture.

A (fig. 43*A*) shows incision at the lateral border of the hard palate through the mucoperiosteum carried beyond the maxillary tubercle and straight out on the buccal mucosa for 1.5 cm., then backward to the level of the lower jaw and then inward, again cutting the pterygomaxillary ligament. The mucosa, pterygomaxillary ligament and the buccinator muscle are cut through and the flap is dissected up until the space between the internal pterygoid and tensor palati muscle is opened. The hamular process is cut through at its base.

The cleft borders of the velum are not freshened in the usual way but the incisions *b-b* (fig. 43*A*) are made on either side through the whole thickness of the soft palate and the flaps behind these incisions are rotated backwards. In this way the incisions *b-b* are opened and the raw surfaces thus exposed are sutured to each other at the median line (see *b*, fig. 43*B*). As the two halves of the velum are carried toward the median line, the flaps *a-a* are drawn inward and there will be no subsequent scar contraction to render the velum tense and comparatively useless. The space between the upper and lower jaw is still cov-

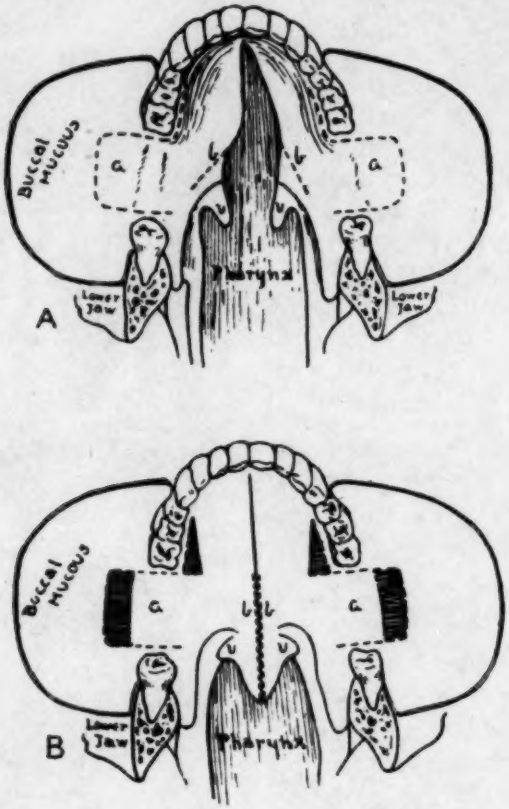

Fig. 43.—Blair's operation. (From Blair.)

ered and opening of the mouth is but slightly interfered with. This operation gives a longer velum than is obtained by the simple Langenbeck operation and therefore a better functional result is obtained. It will not permanently cripple the action of the superior constrictor muscle of the pharynx.

Pickerill,[84] in 1912, developed a procedure by which surgical measures were combined with prosthesis to correct anteroposterior shortening of the palate when the shortening was the result of scar

84. Pickerill, H. P.: New Zealand M. J. **11**:125, 1912.

contraction following a cleft palate operation. The method consisted in altering the cleft velum into a muscular bridge running across the roof of the mouth. This was accomplished by freshening the velum on one side of the cleft and applying a side incision into the velum running anteriorly through its posterior border as shown by A and B in the figure 44 *A*. The other side of the cleft was prepared by freshening its posterior border C and a similar lateral incision was made, as represented by D. These flaps were swung across the roof of the mouth and united with suture so as to obtain the muscular bridge as shown in figure 44 *B*. The sutures were removed on the tenth day. Two

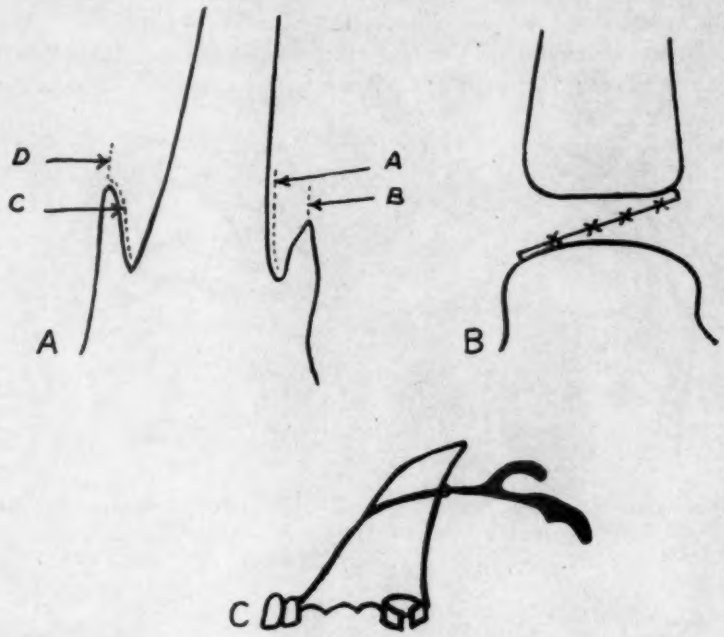

Fig. 44.—Pickerill's method of combining palatoplasty with prothesis: *A*, dotted lines, the margins to be pared and the lateral incisions; *B*, flaps running across and sutured in position; *C*, diagram of appliance used to fill up the cleft, showing the hook by which the hinged velum is attached to the muscular bar and also an artificial vomer attachment.

weeks later the obutrator-velum shown in figure 44 *C* was constructed. The velum of this appliance was made of hard vulcanite with a hook placed on its nasal aspect. The velum was attached by a platinum hinge to an obturator plate fashioned to cover the defect in the hard palate and attached to the teeth. The obturator plate could be fitted with an artificial septum in cases in which the septum naris was absent. The principle of this appliance was such that the hook rested

on the muscular bridge which raised the velum and depressed it as the muscles were brought into play. The velum was also embraced by the superior constrictor muscle of the pharynx during phonation and deglutition.

Naegeli,[85] in discussing Pickerill's method in 1924, stated that three patients were treated in Garré's clinic with marked success.

In my opinion, this operation is impracticable. From my experience in treating patients with cleft palate, it is better to select either a purely surgical procedure or else fit the patient with an obturator-velum. Either one of these methods, when properly applied, will produce good speech. Pickerill's method destroys and deforms, by scar formation, a velum which would have been useful in treating the cleft by ordinary operative procedures. The use of the obturator-vela is difficult in children on account of their growth and dentition. If, for any reason, a child leaves

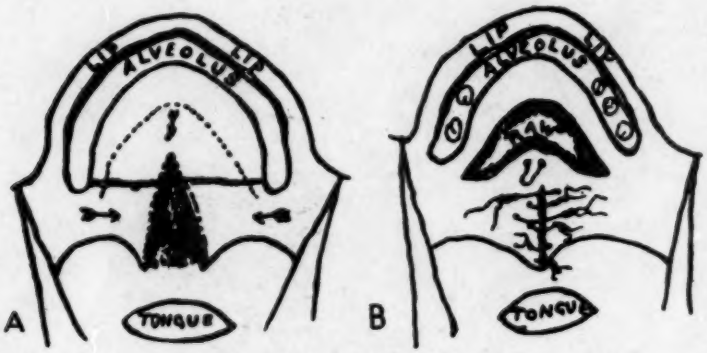

Fig. 45.—Roberts' operation for lengthening the velum. *A* indicates the "apron" flap for lengthening short velum or closing clefts, using intranasal suture; *B*, "apron" flap and intranasal suture. (From Roberts.)

his appliance out of his mouth for several days, it may be impossible for him to reintroduce it as the teeth frequently undergo such a marked change in their position.

Roberts,[36] of Philadelphia, in 1918 published the report of an operation by means of which he produced backward displacement of the palatine mucoperiosteum which remained in continuity with the velum to secure velopharyngeal closure, in cases in which the palate appeared to be lacking in material or was shortened or stiffened. Roberts made a curved incision across the palatine vault in the region of the canine teeth (fig. 45 *A*) and elevated the mucoperiosteum from the underlying bone, freeing the velum from its attachment to the palate bones. This procedure furnished a mucoperiosteal apron suspended from the

85. Naegeli, T.: Schweiz. med. Wchnschr. **5**:62, 1924.

hamular processes (fig. 45 *B*). The velum was then displaced backward like a sling and held by a looped suture passed through the united palate, the ends of which were passed out through the nostrils, where they were held around a rubber tube at the columella (fig. 46).

Veau and Ruppe [86] of Paris, in 1922, called attention to the fact that the classic "Baizeau-Langenbeck-Trélat" procedure for performing cleft palate operations left a denuded area on the nasal surface of the palate, which on healing produced extensive scar tissue, which on retraction prevented the velum from approximating the posterior pharyngeal wall. These surgeons insisted that separate suturing of the nasal mucoperiosteum, the muscular layer of tissue forming the velum by silver wire suture, and the oral muscoperiosteum prevented this undesirable scar contraction. In order to accomplish this, the nasal

Fig. 46.—Nasal suture holding palate flap in position against bone, shown threaded and tied to rubber tubing at nares. (From Roberts.)

and oral muscoperiosteum must be thoroughly separated from the bony palate. They also advised the use of the V-shaped incision introduced by Ganzer [87] in order to obtain the retrotransposition of the palate.

Rosenthal,[78] in 1924, revived Schoenborn's velopharyngoplasty, which he combined with a modified Langenbeck uranoplasty, for all cases requiring closure of the cleft which involves the hard and soft palate. This combined operation was done at one sitting so as to correct the speech deficiency which so commonly followed ordinary cleft palate operation. In performing Langenbeck's uranoplasty, Rosenthal makes lateral incisions as close to the alveolar border as possible. The incision

86. Veau, Victor, and Ruppe, Charles: Rev. de chir. **60**:81, 1922; J. de chir. **20**:1, 1922; J. de méd. et chir. prat. **95**:685, 1924.

87. Ganzer, Hugo: Berl. klin. Wchnschr. **57**:619, 1920.

extends well behind the last molar tooth, where its direction now follows the pterygomandibular ligament, over which it extends to a point 1 cm. from the last mandibular molar tooth (figs. 47 and 48). This method of extending the lateral incision was first used by Blair [35] of St. Louis in 1911.

In February, 1925, Ernst [88] criticized Rosenthal's method, and he suggested circular narrowing of the pharynx so as to permit the velum to approximate the pharyngeal wall. Ernst's article contained no illustrations, and it was impossible to make an adequate mental picture of his operation. Halle,[89] in May, 1925, gave further details of this operation, which is now known as the Ernst-Halle method. The operation is a modified Langenbeck procedure, in which the following steps are added. After freshening the edges of the defect the nasal mucosa is separated from the oral mucosa, the lateral incisions are extended well behind the last maxillary molar tooth, then directed backward and downward so that the incisions terminate in the palatopharyngeus arch, mesial to the last molar teeth (fig. 49 *A*). An elevator is then introduced in such a manner that it remains in intimate contact with the underlying bone, thus avoiding any injury to the palatal muscles. It proceeds behind the superior constrictor muscle of the pharynx, as shown in figure 49 *B*. The space thus formed by the separation behind the pharyngeal wall is then packed with iodoform gauze so as to bring about mesial displacement of the lateral walls of the pharynx (fig. 49 *C*). These gauze packs are changed from time to time until the cavity is well filled with granulation tissue. While waiting for the granulation tissue to form, the anterior attachment of the palatine mucoperiosteum is gradually separated behind the incisor teeth by small incisions. As soon as sufficient collateral circulation is established, the anterior attachment of the palatine mucoperiosteum is completely divided, as shown in figures 49 *B* and *C*. This permits the entire soft tissue of the palate to become displaced backward. The anterior portion of the displaced palate is then sutured at the incisor teeth so as to prevent too much backward displacement. This procedure secures from 1.5 to 2 cm. narrowing of the pharynx, as reported by Halle in cases of incomplete cleft palate. When dealing with cases of complete cleft palate, the V-shaped incision of Ganzer [87] is used, as shown in figures 49 *D*, *E* and *F*. It appears to me that the Ernst-Halle operation cannot be used in children without accepting a high mortality. The V-shaped incision of Ganzer [87] would appear to be ideal, theoretically. Practically, however, when one remembers that the palatine mucoperiosteum is not flexible, one readily

88. Ernst, Franz: Zentralbl. f. Chir. **52**:464, 1925.

89. Halle: Verhandl. d. Gesellsch. deutsch. Hals-, Nasen- u. Ohrenh., May 28, 29 and 30, 1925, Ztschr. f. Hals-, Nasen- u. Ohrenh. **12**:277, 1925.

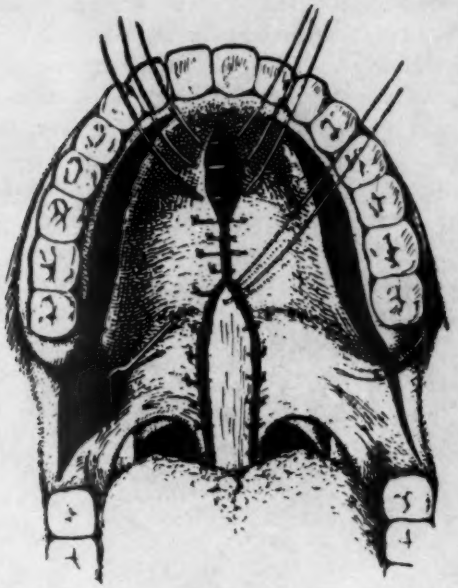

Fig. 47.—Oral view of pharyngeal flap and lateral incisions. (After Rosenthal.)

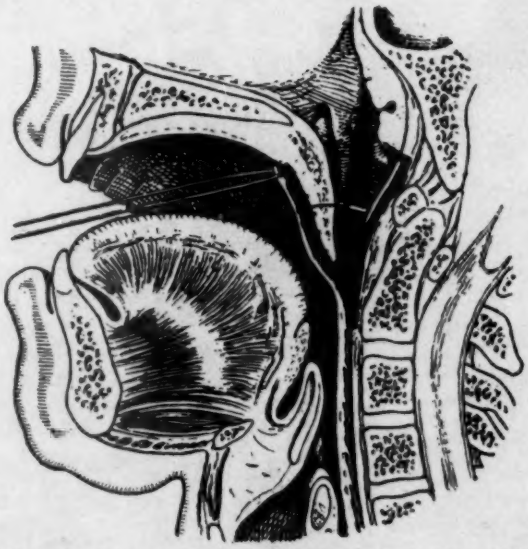

Fig. 48.—Sagittal section to show the pharyngeal flap turned downward to fit into the cleft in the velum. (After Rosenthal.)

realizes that when it is sutured under tension it is bound to tear apart and resume its original position. It has been my experience in plastic surgery that stiff tissue like that of the palatine mucoperiosteum cannot be stretched evenly. By following Ganzer's method in palatine operations, unquestionably puckering of the mucoperiosteum must result.

Von Gaza,[90] in 1926, advised the implantation of fat and fascia tissue into the retropharyngeal space in order to produce bulging of the

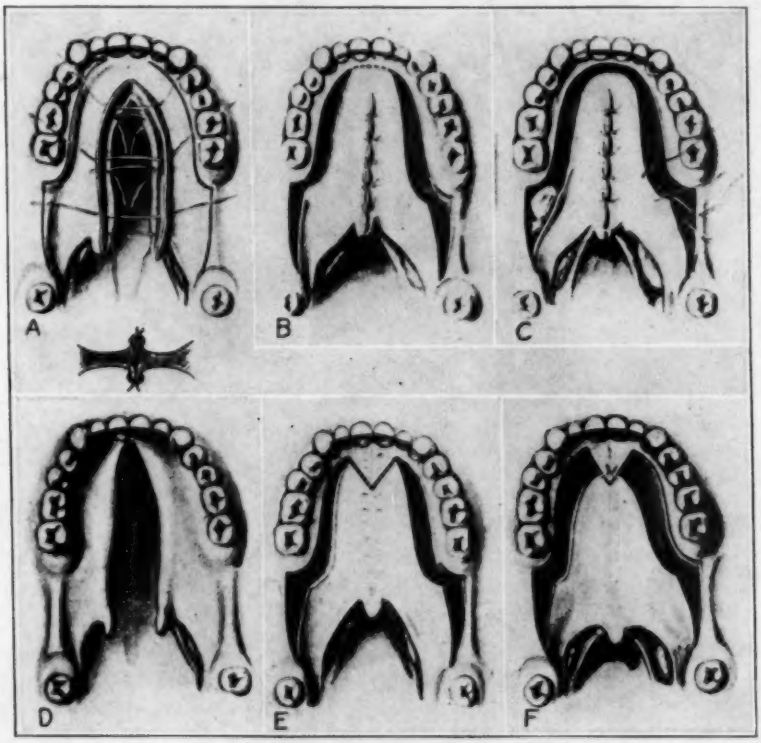

Fig. 49.—In *A*, the upper figure shows the edges of the defect slit open to separate nasal from oral mucosa; the lower figure shows the sides of the defect approximated with the oral and nasal mucosa sutured separately. *B* shows the defect sutured. The dotted line indicates the incision used to separate the anterior attachment of palatine mucoperiosteum. In *C*, retropharyngeal packing is seen on the right side and the retaining sutures on the left side. *D* shows the cleft palate extending as far anteriorly as the alveolar margin. In *E*, the palate is completely closed in the midline. The V-shaped incision of Ganzer is shown. *F* shows how Ganzer's V-shaped incision is held anteriorly to the mucoperiosteum behind the incisor teeth. (After Halle.)

pharyngeal wall. He condemned the intra-oral method of approach because of fear of infection, and suggested entrance through the neck

90. Von Gaza, W.: Arch. f. klin. Chir. **142**:590, 1926.

by way of the superior lateral triangle. He obtained the fat and fascia from the abdomen or gluteal region, except in children for whom he advised the use of the fascia lata. The operation consists of making a curved incision from 3 to 5 cm. in length, dividing the skin, superficial fascia and platysma muscle, the incision being made one fingerbreadth from the mastoid process just below the lobe of the ear and in front of the anterior border of the sternocleidomastoid muscle (fig. 50). The incision extends as far down as the angle of the mandible. The tissue behind the jugular vein is removed by blunt dissection. Entrance into the retropharyngeal space (figs. 51, 52 and 53) is made by proceeding inward, forward and upward, using as a guide the tendons of the longus capitis and longus colli muscles which lie anterior to the vertebral column at the level of the third cervical vertebra. The pharyngeal wall is pushed forward by the finger and separated from the prevertebral tissues on both sides. The cavity formed by this separation is then

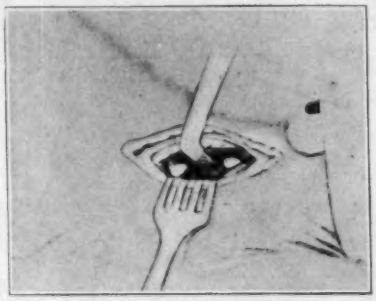

Fig. 50.—Incision exposing the cervical glands with the vascular and nerve bundles drawn aside. The longus colli muscle is exposed. (After von Gaza.)

temporarily packed with gauze to control bleeding. Later, the gauze is replaced by the transplant. Von Gaza mentioned the fact that Perthes suggested the use of cartilage transplants into the retropharyngeal space for the correction of velopharyngeal insufficiency. The operation, in the hands of von Gaza, appears to be a simple, bloodless procedure. In my opinion, however, it calls for a great deal of skill and is not without danger. When the operation is performed on children, it has one point in its favor in that it will free the superior constrictor muscle of the pharynx, thus permitting it to come forward. Nevertheless, when a graft is introduced into the retropharyngeal space in a child the implanted tissue will not grow in size; in fact, it will tend to shrink, whereas the pharyngeal canal will increase in size. I feel that the method is not justified in the average case, as Passavant's cushion can be increased in size by such simple procedures as massage, electricity and speech training.

Kirkham,[91] in 1927, pointed out that in cases of cleft palate the distance between the hamular processes is greater than in normal mouths. He further stated that the increase is in proportion to the width of the cleft. In his opinion, velopharyngeal insufficiency is due more to the widened condition of the pharynx than to the shortened state of the velum. Kirkham reached his conclusion after making measurements of normal specimens of the palate and comparing these with specimens of cleft palate of the same age at the Hunterian Museum of the Royal College of London. Basing his operation on these observations, Kirkham shortened the superior constrictor muscle of the pharynx

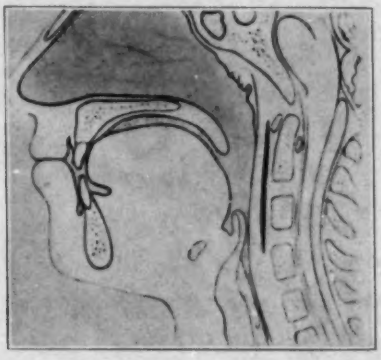

Fig. 51.—Sagittal section, showing retropharyngeal space to receive fat tissue. (After von Gaza.)

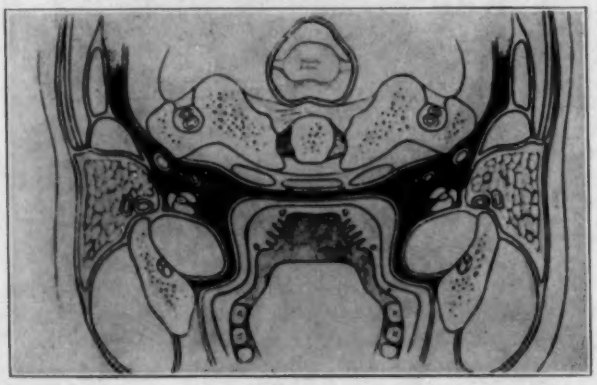

Fig. 52.—Horizontal section at the level of the second cervical vertebra, showing loose tissue behind the pharyngeal wall. The dark area shows areolar tissue. (After von Gaza, modified from Corining.)

91. Kirkham, H. L. D.: Surg. Gynec. Obst. **44**:244, 1927.

so as to permit it to contract more forcibly. The operation consisted of freshening on either side the upper edge of the superior constrictor muscle and suturing this to the corresponding freshened side of the palatopharyngeus muscle (fig. 54). This operation was performed by Kirkham in a boy 6 years of age and was a complete failure.

Woods,[92] in 1927, published an article in which he reported success with the injection of paraffin into the retropharyngeal space in cases in which the velopharyngeal opening remained moderately small after a standard cleft palate operation. He also used a method analogous to that of Rutenberg, which he describes in the following words:

> In 1925 I was consulted by a lady, age 30 years, on whom quite a good operation had been performed but the opening behind the palate was literally huge. She felt her disability quite acutely and was most anxious that something should be done. I consented to try. I thought by snipping away the mucous membrane of the nasopharynx at the level of the palate, so as to form a linear wound there might be some hope that, after cicatrization when the scar contracted, the defect might be so reduced that she could completely cure it by training the muscles. I accordingly snipped away a ring of tissue but the result was most disappointing as the remedy proved quite futile. The wound healed with the most aggravating rapidity, leaving her neither better nor worse for the attempt.

Fig. 53.—Roentgenogram showing a gold thread placed in the transplanted fat tissue. The pharyngeal wall has been brought forward. (After von Gaza.)

This failure was followed by the use of diathermy. From the latter procedure, Woods claimed a fair amount of success. From Wood's description of the application of diathermy, one is led to believe that he meant electrocoagulation. He describes the treatment in these words:

> I next tried diathermy. This produced a slough of the superficial tissues of the sides and back of the pharynx and the edge of the soft palate, including the

92. Woods, Sir Robert: Brit. M. J. **1**:371, 1927.

uvula. The throat was very sore and painful for some days. She had no rise in temperature. After a week or so the wound cleared up and contraction began. This continued until about three months later. The wound was reduced to the size of the tip of the small finger. I found then that it was quite easy for her to close it completely by an effort of the will. She thus had all the physical conditions necessary for normal speech.

Limberg [18] of Leningrad, in 1927, criticized the Ernst-Halle procedure for obtaining retroposition of the palate, this criticism being directed at the fact that they divided the posterior palatine vessels and

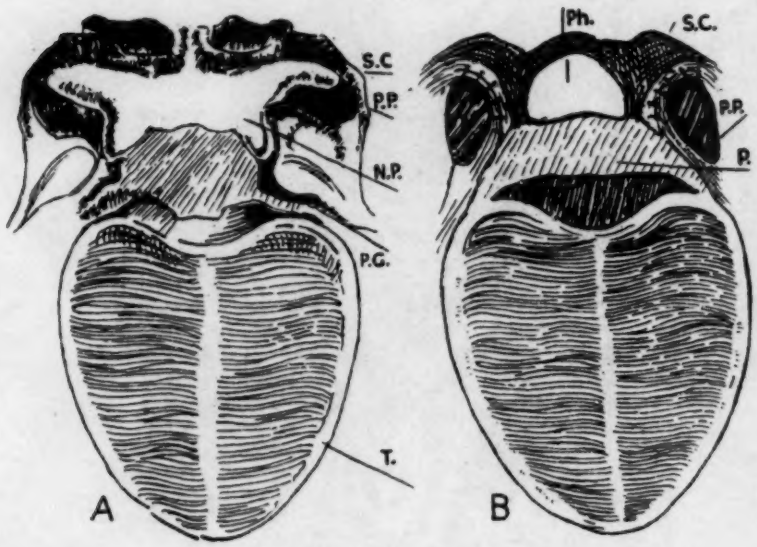

Fig. 54.—*A* is a cross-section of the nasopharynx just above the level of the uvula. *S.C.* indicates the superior constrictor; *P.P.*, palatopharyngeus; *N.P.*, nasopharynx; *P.G.*, palatoglossus; *T.*, tongue. *B* shows diagrammatically a cross-section of the point where the superior constrictor was sutured to the posterior surface of the palate and the palatopharyngeus muscles. *Ph.* indicates the pharynx; *S.C.*, superior constrictor; *P.P.*, palatopharyngeus; *P.*, palate. (From Kirkham.)

nerves. To preserve the continuity of these structures and to secure complete relaxation of the soft tissues of the palate with its backward displacement, Limberg proposed two formidable operations. The first of these methods consisted of resection of the greater palatine foramen and interlaminar osteotomy of the pterygoid process. The second procedure was accomplished by pterygomaxillary osteotomy.

Resection of the greater palatine foramen was accomplished by means of the chisel or bone-cutting forceps, cutting mesially and posteriorly to the foramen so as to release the vessels and nerves and displace them inwardly and posteriorly (fig. 55).

Interlaminar osteotomy of the internal pterygoid plate is performed by placing the chisel on the palate bone external to the greater and lesser palatine foramina. The cutting edge is directed parallel with the perpdendicular plate of the palate bone in such a manner as to resect the internal pterygoid plate from the external at their point of articulation with the palate bone. This resection permits mesial transposition

Fig. 55.—*I* shows how interlaminar osteotomy is made; *II*, how pterygomaxillary osteotomy is made. (From Limberg.)

of the mesial pterygoid plate with its hamular process, and keeps the anatomic continuity of the vessels and nerves intact (fig. 55). Pterygomaxillary osteotomy was accomplished by introducing the chisel through the horizontal plate of the maxilla between this bone and the pterygoid plate. The process was then separated and moved inward toward the mesial line. While Limberg preserves the relation of the posterior palatine vessels and nerves, as has been already pointed out, all surgeons agree that it is impossible to liberate the palatine mucoperiosteum and secure the desired backward displacement without injury to these vessels and nerves. From my experience in the operating room, and from postmortem studies of operative cases, as well as anatomic studies,

I am convinced that the nerves and vessels are usually divided during cleft palate operations. I further believe that no injury results to the patients from the division of these structures.

In May, 1928, Moorehead,[93] of Chicago, published an article in which he described an operation for correcting secondary palatine defects by using lateral incisions similar in principle to those used by Ernst-Halle and by myself [94] for gaining retrotransposition of the velum to correct cleft palate speech. The V-shaped incision described by Moorehead was first suggested by Ganzer [87] before 1920 and later modified by Ernst and Halle in 1925 (fig. 56).

In 1928, Wardill [95] developed a two-stage operation by which he combined narrowing of the pharyngeal canal with closure of the existing cleft in the palate to establish a good speaking voice. The first stage

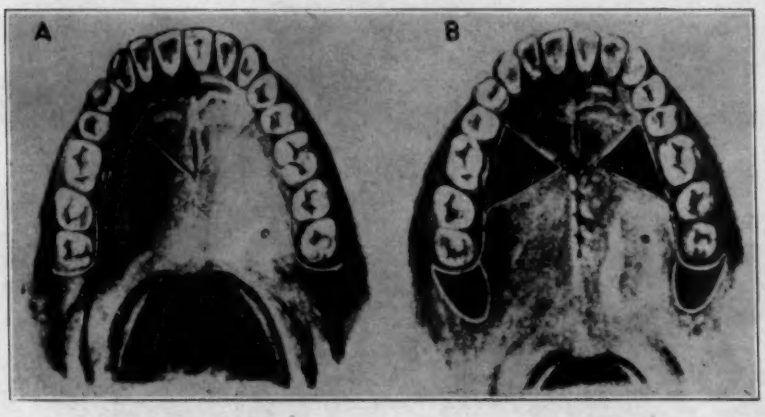

Fig. 56.—Method of transposing the palate to: (*A*) lengthen the palate and (*B*) release tension and make possible the closure of the nasopharynx in speech. (After Moorehead.)

of this operation is analogous to that of Rutenberg and consists in narrowing the nasopharynx laterally with the production of a more prominent Passavant cushion. Wardill described this step of the operation in the following words:

> The patient, lightly anesthetized, is laid upon the back with a pillow beneath the shoulders and the head thrown well backwards. The mouth is held open with a gag. The pharynx is stimulated and the position of the ridge of Passavant is noted. With a fine tenotome, the mucous membrane is incised trans-

93. Moorehead, F. B.: Correction of Secondary Plate Defects, J. A. M. A. **90**:1614 (May 19) 1928.

94. Dorrance, George M.: Ann. Surg. **82**:208, 1925; J. Am. Dent. A. **14**: 1112, 1927.

95. Wardill, W. E. M.: Brit. J. Surg. **16**:127 (July) 1928

versely at the level of the ridge of Passavant, i. e., over the anterior arch of the atlas, and is then held open with fine sharp hooks (fig. 57*A*). Care is taken not to incise too deeply and thus open the loose areolar tissue between the buccopharyngeal and prevertebral layers of fascia. The buccopharyngeal fascia is relatively tough, and the superior constrictor muscle and its covering mucosa can be readily peeled or scraped from it by means of blunt dissection with a curved aneurysm needle. The dissection is carried on in this layer, upwards nearly as far as the base of the skull, downwards for a similar distance, and laterally beyond the ridge caused by the salpingopharyngeus muscle. During the whole of this part of the operation some resistance is felt to the dissection. If this resistance is not felt, it is probable that the point of the needle is in the loose retropharyngeal cellular tissue.

Using the aneurysm needle as a combined director and retractor, the incision is enlarged laterally right up to the salpingopharyngeus by the use of a curved

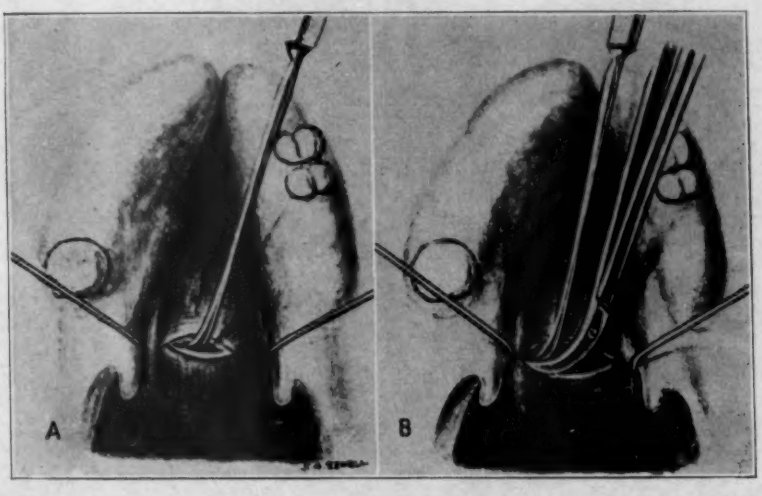

Fig. 57.—*A* shows separation of the superior constrictor from the buccopharyngeal fascia; *B*, extension of the incision as far as the salpingopharyngeus. (From Wardill.)

scissors (fig. 57*B*). Bleeding is very small in amount, there being no more than a slight ooze. The business of sewing up the incision in a vertical direction is now undertaken. I have had a small spring wire retractor made (fig. 58) for the purpose of facilitating this procedure. The retractor holds the incision open in a vertical direction. Using very small curved needles made on the Mayo principle, sutures of No. 0 catgut are inserted, commencing first at the upper and lower ends of the wounds, the sutures are left long and may be used as retractors. It is important to take big bites of tissue on each side, as the sutures have a tendency to work out very quickly. The suturing is completed in the center of the wound by joining together the salpingopharyngeal folds, including a sufficient bite to obtain a firm hold of the tough connective tissue which is intimately bound up with these (fig. 59). Usually a suture is passed through the posterior pillars of the fauces for the purpose of relieving the tension for a few days. At

the end of the operation the halves of the soft palate are found to be much closer together, and the halves of the uvula may overlap. In fact, one wonders how the patients are going to be able to swallow.

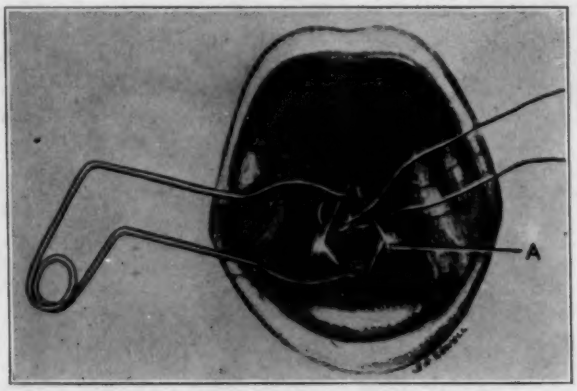

Fig 58.—Suture of the wound in a vertical direction. *A* shows the edge of the salpingopharyngeal fold. (From Wardill.)

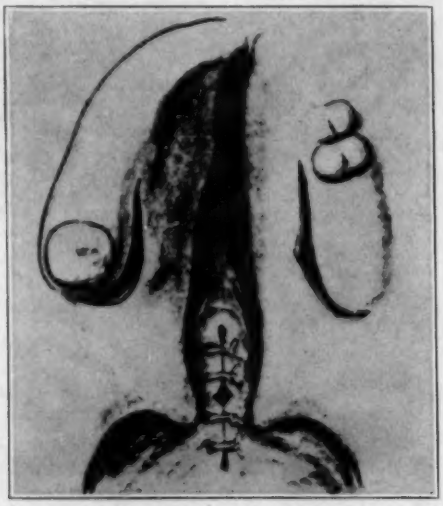

Fig. 59.—First stage of the operation completed. Lateral incisions were made for Langenbeck-Fergusson repair. (From Wardill.)

CONCLUSION

Several years ago, I was confronted with a case of congenital short palate, a condition for which no known treatment had succeeded in obtaining normal speech. I visited many of the well known clinics

of Europe and the United States. The consensus among cleft palate operators appeared to be that where a long velum was present excellent results could be obtained with any standard cleft palate operation, i. e., the Langenbech operation or any of its modifications.

All agreed that failures in cases occurred in which velopharyngeal closure was insufficient. The failures in these cases consisted chiefly in speech defects. The patients could not speak any better after the operation than before. The inability to correct the speech deficiency in congenital insufficiency of the palate was the reason for this study.

A complete survey was next made of all methods employed for correcting this defect. I was dissatisfied with all the methods that had been suggested. No operation appeared applicable in every case, and many of these operations were useless.

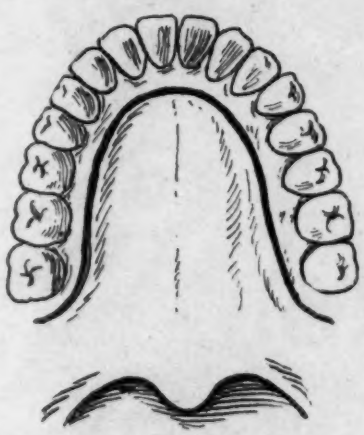

Fig. 60.—Incision running parallel with the alveolar margin, and as near to it as possible, to liberate the palatine mucoperiosteum, used in the "push-back-operation."

Guided by past experiences, I felt that many useful suggestions were made by former authorities and forgotten by their successors. This has been the case in practically every surgical subject. Hence a complete survey of the literature on congenital insufficiency of the palate was undertaken, a résumé of which appears in the body of this paper.

As outlined in the essay, three methods of treating velopharyngeal insufficiency were employed either alone or in combination.

1. The use of prosthetic appliances in the form of obturator-vela.
2. Operations to produce constriction of the pharyngeal canal.
3. Operations to lengthen the velum.

Most patients with a congenital shortening of the palate cannot tolerate any contrivance within the mouth. Irritation is produced, spasms occur, and alterations in the apparatus must be made constantly to obtain a satisfactory result.

The following methods suggested for the constriction of the pharyngeal canal may be considered:

1. Those obtained by cauterization.
2. Those obtained by plastic operation.
3. Those obtained by the implantation of tissue or paraffin in the retropharyngeal space.

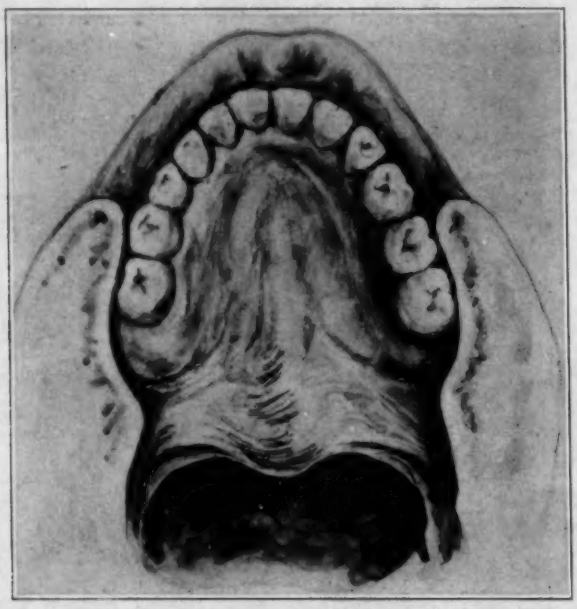

Fig. 61.—Artist's presentation of a case of congenital shortening of the palate.

Cauterization can be dismissed as useless, as it produces a marked amount of scar tissue, interferes with the function of the pharynx, disturbs the normal function of the pterygopharyngeus muscle and leads to improper ventilation of the middle ear.

Operations on the pharyngeal wall were condemned because better results can be obtained by methodic speech training, as this causes hypertrophy of the superior constrictor muscle of the pharynx and permits the pterygopharyngeus portion of the superior constrictor muscle to assist the velum in securing velopharyngeal closure.

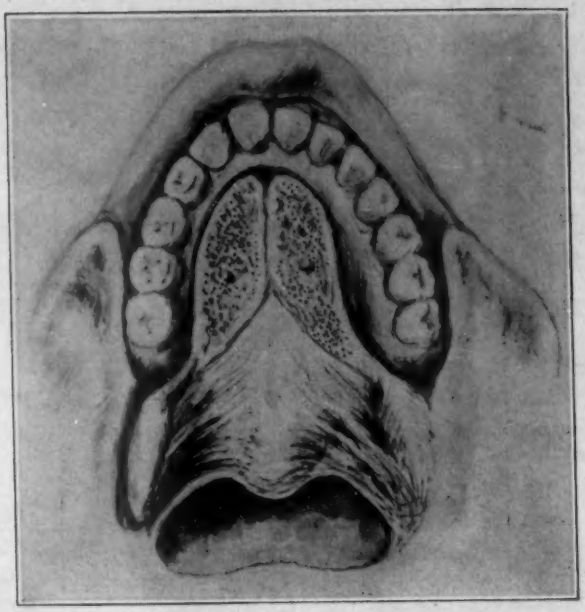

Fig. 62.—Artist's presentation of the case in figure 61 with congenital shortening of palate with the mucous membrane removed.

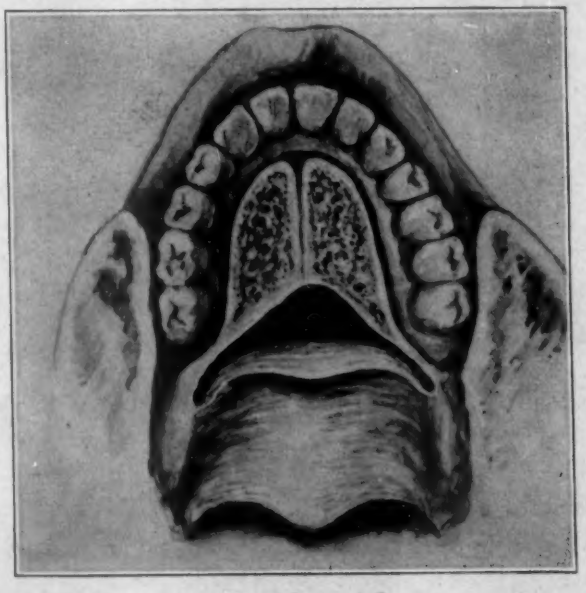

Fig. 63.—View of case in figure 61 after the mucoperiosteum is elevated and the palate completely freed from its attachment to the palatal processes. The fan-shaped portion of the tensor palati muscle is cut.

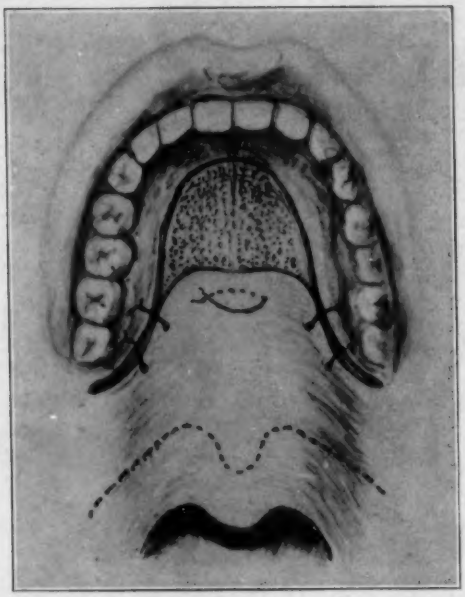

Fig. 64.—Case of congenital shortening of the palate shown in figure 61 after operation. Compare with the original and note the lengthening of the velum secured. The dotted line shows position of palate before operation.

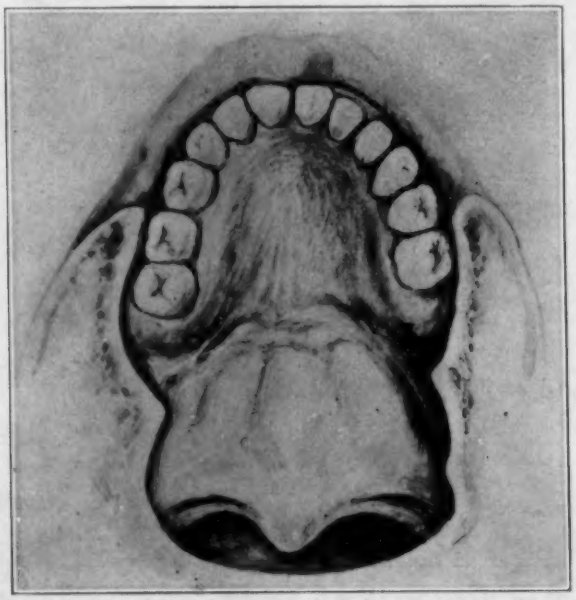

Fig. 65.—Case of congenital short palate after complete healing from the "push-back-operation."

While forward bulging of the pharyngeal wall can be produced by the injection of paraffin or the implantation of fat and fascia into the retropharyngeal space, the first of these methods is so uncertain, as the material injected does not stay in the desired position, and the latter method is fraught with danger which is out of all proportion to the possible benefits to be obtained.

Lengthening of the velum by plastic operations was practically always a failure, since the operators failed to realize that the palatine attachment of the levator palati muscle is placed too far anteriorly and the tensor palati muscle is shorter in all cases of insufficiency of the palate. Hence, it does not make any difference how much the levator palati muscles pull, they cannot possibly bring the velum into its normal position to secure velopharyngeal closure.

While Billroth's method of dividing the hamular process and Agnew's method of cutting the tensor palati muscle relax the tensor palati muscle, these procedures do not change the insertion of the levator palati muscle.

Division of the tensor palati, the palatoglossus and palatopharyngeus muscles, as suggested by Mears, does not in any way displace the position of the insertion of the levator palati muscles.

I spent considerable time in the anatomic department working out various methods of lengthening the velum.

On dissecting a specimen with a cleft palate, I realized the important fact that the insertion of the levator palati muscle is placed so far forward that it could not pull the velum against the posterior pharyngeal wall as the normally placed muscle could. Realizing this fact, the solution must be to devise some means of displacing the palate backward so that the levator palati could normally perform this function. I was then struck by the fact that when the elevated palatine mucoperiosteum was completely freed from its attachment to the bony palate the fan-shaped portion of the tendon of the tensor palati which is inserted into the palatine aponeurosis prevented backward displacement of the flap. Division of this fan-shaped portion, along with freeing its attachment to the bony palate, permitted the palate to fall backward by its own weight so that it approximated the pharyngeal wall. This backward displacement of the velum places the levator palati muscles in a position which approaches the normal. I then devised the following operation:

An incision is made as outlined in figure 60. The flap is freed from the bony palate from before backward (fig. 62). Dissection is carried down to the attachment of the palatine aponeurosis at the posterior edge of the bony palate (fig. 63). The posterior palatine arteries are divided in this operation. This constitutes the first stage of the operation. In some cases it has been possible to do the operation

in one stage, when it was felt that the blood supply was adequate. However, as a general rule, the two-stage operation is more satisfactory. The flap is replaced to its original position and the edges sutured. Ten days later, the sutures are removed and the flap raised again. The attachment of the palatine aponeurosis to the bony palate is divided, and that portion of the tendon of the tensor palati muscle which is inserted into this aponeurosis is also divided (fig. 64). The flap will now fall backward by its own weight against the posterior wall of the pharynx. The anterior edge of this flap is then sutured to the hard palate and to the soft tissue on either side as illustrated in figure 64. In addition to this, the flap is further supported by placing an appliance on the teeth to support the flap. Silver wire passed around each of the molar teeth may be substituted for the appliance. The denuded surface caused by the posterior displacement of the velum rapidly fills in with granulation tissue (figs. 64 and 65). I have used this simple method with success in three cases of congenital insufficiency of the palate and in seven cases of shortened palate in which operation had previously been performed either by myself or by other surgeons, but in which velopharyngeal closure was insufficient. The rationale of this method is based on the principle of surgery that holds elsewhere in the body, that muscles must be placed in as near a normal position as possible, in order to obtain their proper function. My patients have been under observation for a sufficient period of time to note the marked improvement in the functional results obtained by this method. With speech training, they can be made to speak almost perfectly.

This research, then, has solved the problem of correcting congenital insufficiency of the palate.

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THE COURSE OF THE NERVE FIBERS TRANSMITTING SENSATION OF TASTE*

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In 1822, Francois Magendie after dividing the trigeminal nerve within the skull in dogs made the following statement: "The question of taste, formerly so obscure, no longer presents any difficulty. Physiological experiments and pathological observations have solved it. If the trunk of the fifth nerve is divided in the skull, taste is completely lost, even for sour and bitter substances. This total loss of taste has been noticed in persons in whom the fifth nerve has been compressed or altered." The problem of taste was not, however, to be settled so simply and unequivocally.

As it has been impossible to expose the nerves concerned in the mediation of taste sensations throughout their courses, indirect methods of study, such as the following, have been employed: (1) clinical studies checked by pathologic observations, (2) experiments on animals and (3) postoperative observations on human beings. From the evidence which has accumulated, one theory remains constant: the rôle of the chorda tympani in the transmission of sensations of taste. All observers admit that it does transmit these sensations, but there is no agreement as to the route of the taste fibers in this nerve from the terminal sense organs to the central nucleus.

At the time Magendie performed his experiments, little was known concerning the function of the chorda tympani. A few years before, Bellingeri (1818) described it as the sensory part of the facial nerve and assumed that it transmitted sensations of taste to the brain. The significance of this assumption seemed, however, to have been little appreciated until Claude Bernard, Magendie's successor, made his contributions to the studies of taste. Montault, a pupil of Romberg, and Bernard presented a thesis (1831), in which three cases of facial paralysis accompanied by loss of taste over the anterior two thirds of the tongue were reported. In 1843, Bernard reported four more cases, in which the facial palsy was caused by acute infections and fractures. He noted that the general sensitivity of the side involved was not affected and that taste lost during the paralysis returned with recovery. Section of

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* From the Johns Hopkins Hospital and University.

the nerve, earlier performed by Magendie, was repeated by Bernard with a knife especially designed for this purpose by Magendie. After division of both facial nerves intracranially he noted loss of taste similar to that which followed section of the chorda tympani in animals. He believed, however, that the loss was not complete, but that the acuity of taste sensation was markedly lessened.

Bernard must have tested for loss of taste after section of the trigeminal nerves in dogs—an experiment frequently made by him—but there is no mention of any such tests in his protocols. He was particularly interested in the trophic changes that followed section of the fifth nerves and these are frequently mentioned, but curiously enough no mention is made concerning the effect of such division on taste. He accepted the views of Magendie, his preceptor, that the fifth nerve transmitted sensations of taste, and sought to explain alterations in taste following division of the facial nerve as due to lack of "motor control" over the taste buds. He stated, "The chorda tympani should be considered a motor nerve, which by its action upon the papillary lingual tissue regulates and renders instantaneous the transference of a rapid stimulus to the sensory nerve which conducts it to its center." When paralyzed, it affects taste, "not as a nerve of sensation, but by a special inertia of the gustatory organs."

It was not then generally conceded that the chorda tympani played an important rôle in the conduction of sensations of taste. Tests which confirmed this view were shortly to be made. Duchenne (1850) and Blau (1879) took advantage of fistulas of the middle ear to make observations on the function of the chorda tympani. They found that by stimulating the internal wall of the tympanum by irrigations, cautery and faradic current, they could produce sensations of taste in the anterior two thirds of the tongue, but not in the posterior one third. At times the sensation so produced was sweet, at other times sour, while at still other times a prickly sensation was produced. Blau stated that Troltsch had previously produced much the same sensations by pinching granulations over the chorda tympani on the internal wall of a chronically inflamed middle ear. Since these experiments were made, ample opportunity has been afforded to study the effect on taste of division of the chorda tympani nerve during operations on the mastoid and middle ear.

Guzot and Cazalis (1839) were credited by Luciani, and Biffi and Morganti (1846) by Lussana with discovering that the chorda tympani carries taste sensations from the anterior two thirds of the tongue only and that the glossopharyngeal nerve serves the same function for the posterior one third. As previously stated, however, Montault appears to have called attention to this dual supply several years before (1831).

It is now generally accepted that the chorda tympani conducts all taste fibers from the anterior two thirds of the tongue, but there is no unanimity of opinion as to the path that these fibers pursue from the geniculate ganglion in the facial to their central nucleus. The attempts to determine the course of these fibers may be assigned to three periods which, in a general way, conform to three types of investigation. The first period, one in which animal experimentation predominates, covers the first two thirds of the nineteenth century. This period was dominated by Magendie, Bernard and Schiff. The second period, largely one of clinical and pathologic studies, supplemented now and again by animal experimentation, covers the remaining one third of the nineteenth century. The third period, beginning a few years before the close of the nineteenth century and extending to and including the present time, is concerned with tests on patients in whom the cranial nerves have been divided intentionally for the relief of painful tic or some definite lesion.

Several different pathways of the fibers of the chorda tympani from the geniculate ganglion of the facial nerve to the central nucleus have been proposed. These proposals have been based on experimental, clinical and operative observations.

PATHWAY PROPOSED BY SCHIFF

Schiff (1867) made a radical departure from the teachings of Magendie and Bernard. We have already noted that it had been conceded by most observers that the chorda tympani contained all the fibers of taste from the anterior two thirds of the tongue. Schiff made the following experiments:

1. He cut the fifth nerve in animals between the brain stem and the gasserian ganglion and found that the sense of taste was lost. He found that if he cut the facial nerve at the pons, taste was not affected, and therefore concluded that the sensations of taste must pass to the brain through the sensory part of the fifth nerve.

2. He found that when he cut the facial nerve at the geniculate ganglion or distal to it the sense of taste was destroyed, and concluded that these fibers must be deflected to the fifth nerve at the geniculate ganglion.

3. He then cut the third branch of the fifth nerve at the foramen ovale without influencing taste, but found that taste was lost when the second branch of the fifth nerve was divided at the foramen rotundum.

He concluded, therefore, that the fibers of the chorda tympani which carried sensations of taste passed by way of the great superficial petrosal nerve from the geniculate ganglion to the sphenopalatine ganglion, by

way of the second division of the fifth nerve to the gasserian ganglion and then through the sensory root to its central nucleus.

The validity of Schiff's experiments and the entire conception of the course of the taste fibers suggested by him were quickly questioned. His results also differed markedly from those of Claude Bernard, who found that taste was profoundly changed after intracranial division of the facial nerve.

However, some early clinical reports appeared to support Schiff's views. Erb reported a case in which the second branch of the fifth nerve was destroyed by a "mass of inflammatory" tissue; Senator

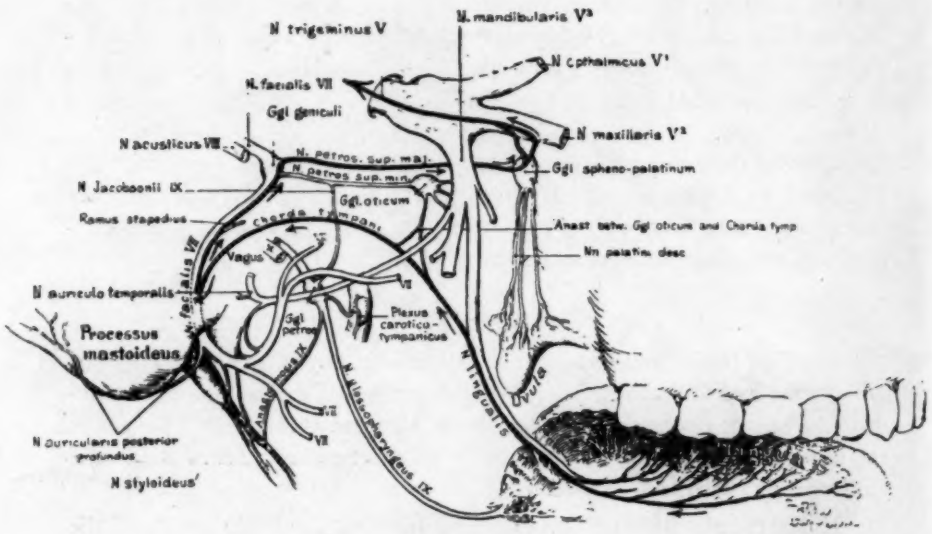

Fig. 1.—Pathway for taste fibers suggested by Schiff.

(1882) and Salomonsohn (1886), cases in which the nerve was involved in a "chronic inflammatory mass in the middle fossa," and Heusner (1886), another case of the tuberculous caries of the bone (but there was no autopsy). In these cases taste was lost over the anterior two thirds of the tongue. These lesions were too diffuse to warrant the conclusions that were made concerning the course of the nerves involved in taste.

For a long time, Schiff had considered it probable that not all the taste fibers passed through the chorda tympani, but that part of the fibers at least passed through the lingual. He thought, however, that these fibers did not pass directly to the gasserian ganglion, but that they were deflected to the otic ganglion, then to the geniculate by the lesser superficial petrosal nerve and back again to Meckel's ganglion by way

of the great superficial petrosal nerve, and by branches from Meckel's ganglion to the second branch of the trigeminus.

LUSSANA'S OBSERVATIONS AND PATHWAY PROPOSED BY HIM

Two years after Schiff suggested the route just described, Lussana (1869) made an impressive contribution to the study of the course of the fibers of taste. He assembled a well selected group of clinical cases, and from a study of these arrived at a new conception of the course of taste fibers. He concluded that the nervus intermedius (Wrisberg) was the only nerve which transmitted sensations of taste from the anterior two thirds of the tongue.

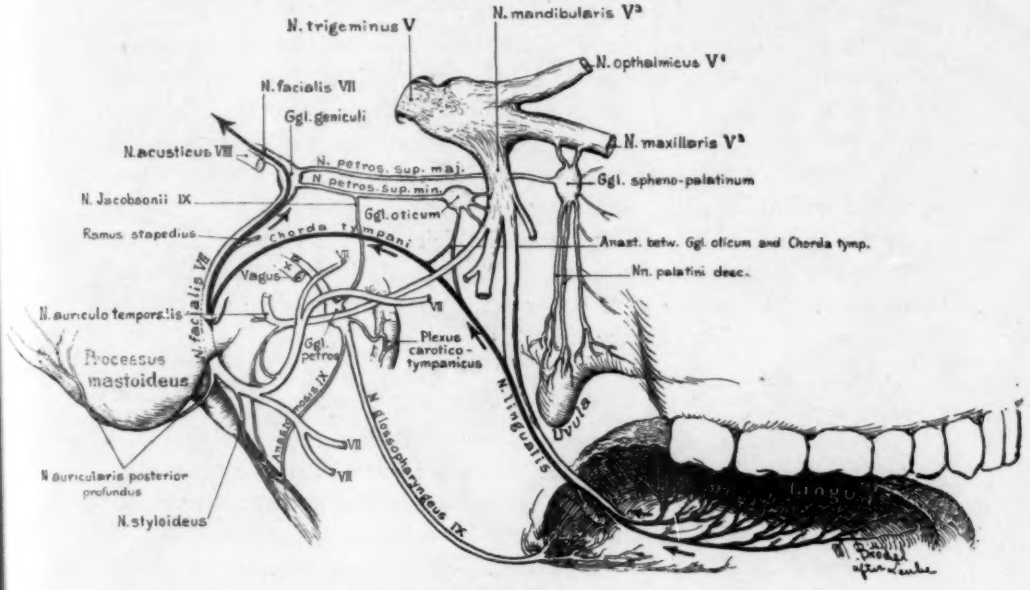

Fig. 2.—Pathway for taste fibers suggested by Lussana.

Lussana's first case was seen with his associate Renzi. The patient had complete loss of sensation over half of the face and tongue (fifth nerve), with no impairment of taste. At autopsy the seventh and eighth nerves were found intact, the fifth destroyed.

His second patient had facial neuralgia. The lingual nerve, including the chorda tympani, had been divided by an operation—"the first time such a procedure had been done in the history of science." Taste was completely lost over the anterior two thirds of the tongue, but pain sense was not affected. This experiment was used by him to eliminate the glossopharyngeal nerve as the nerve of taste for the anterior two thirds of the tongue.

His last and crucial case was again "the only case of its kind in science." In this patient the chorda tympani had been divided, and there had been no injury to the fifth nerve. The patient, a man aged 48, had gradually become deaf in one ear, and in seeking relief had fallen into the hands of a charlatan who, in the presence of many onlookers, attempted to demonstrate his skill. Three times a long, narrow lance was thrust deeply into the ear. The patient fell to the floor in great agony, a convulsion followed and hemiplegia developed. The charlatan fled in haste from the angry mob. Lussana found this patient two years later. The paralysis had cleared, sensation in the face was normal, but taste for the anterior two thirds of the tongue was still lost. The chorda tympani had been divided.

Lussana supported his unique clinical observations by experiments on animals. After cutting the inferior maxillary nerve above the entrance of the chorda tympani into the lingual or intracranially, he found no alteration in taste. When the chorda tympani was divided in the tympanum, taste was permanently lost. Three years later he published additional data supporting his original contention and referred to the "remarkable" earlier work of Stich (1851), who had collected from the literature many cases of facial paralysis accompanied by loss of taste. Lussana's earlier paper provoked much adverse criticism on the part of Schiff, Vulpian and Vizioli.

PATHWAY SUGGESTED BY ZIEHL

Ziehl (1889) proposed still another pathway, thus adding to the confusion which already existed. He too believed that the taste fibers entered the brain by way of the fifth nerve, but thought that they passed by way of the third branch into the gasserian ganglion instead of by the second as Schiff had suggested. This course was based on the observations of Romberg, confirmed by autopsy which was "performed by Henle in the presence of Johannes Müller." In this case taste was lost over the anterior two thirds of the tongue on one side, and at autopsy a mass of granulation tissue was found which compressed the third division of the fifth nerve at the foraman ovale. Ziehl believed that the observations in this case indicated that sensations of taste were carried to the brain by the third branch of the fifth rather than by the second as thought by Schiff and Erb. Accepting the view that all taste fibers pass up the chorda tympani to the geniculate ganglion, he assumed that they then passed by way of the small superficial petrosal nerve directly or by detour through the tympanic plexus, probably in both ways, to the otic ganglion and then by its communicating branches to the third division of the fifth through the gasserian ganglion to the brain.

Many of Ziehl's views were based on the none too definite results of animal experiments made by Vulpian, who claimed that taste was lost after the fifth nerve was divided intracranially, but was unaffected when the seventh and ninth were divided within the skull.

Ziehl's theory was the one that later become most generally accepted. It received support from a new line of evidence: the effects on taste of operations on the gasserian ganglion performed by Krause. Ziehl's theory also seemed to be supported by a case reported by Ferguson, who found at autopsy in a patient in whom taste to the anterior two thirds of the tongue had been lost, an exostosis which compressed the vidian nerve.

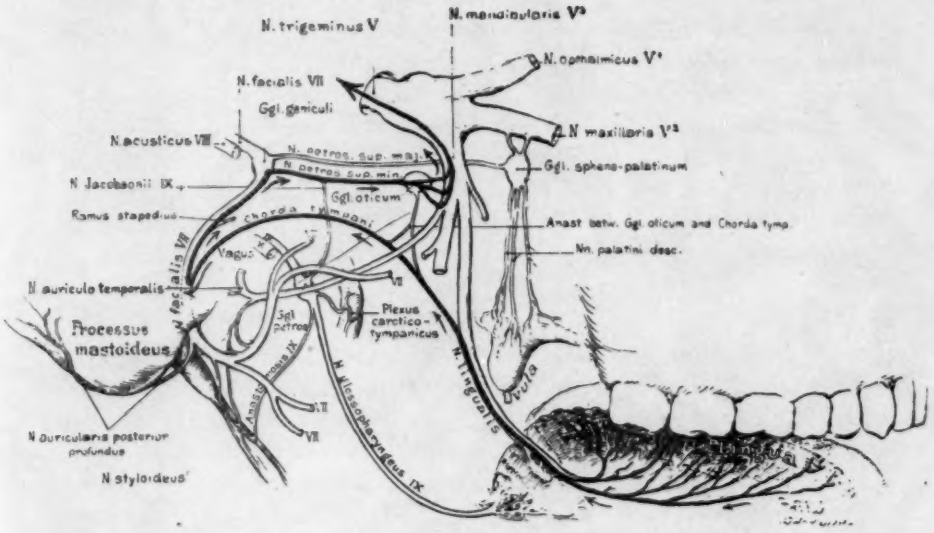

Fig. 3.—Pathway for taste fibers postulated by Ziehl, and later supported by the operative results of Krause on human subjects.

Gowers (1885 and 1897) ardently championed the theory that the fifth nerve was the pathway, without indicating preference for either the second or third branch. He not only accepted the circuitous pathway proposed by Schiff, but, as the result of the examination of three patients in each of whom taste was lost over the entire half of the tongue, was convinced that "taste impressions reach the brain solely by the roots of the fifth nerve and that the doctrine that the roots of the glossopharyngeal nerve had anything to do with taste is a curious myth, due to too wide an induction from certain anatomic facts and from dubious experiments on animals." And again, "It is possible that nerve fibers for taste on the back of the tongue may be distributed with the

ninth nerve, reaching there from the otic ganglion of the trigeminus by the small petrosal nerve and the tympanic plexus. This course, I confess, seems strangely circuitous, but it is scarcely more circuitous than that which is certainly taken by taste fibers of the front of the tongue." Although Gowers' publications were twelve years apart, his views on taste remained unchanged. However, despite Gowers' standing in the medical world, his claim that the fifth nerve conducted taste fibers from the posterior one third of the tongue gained few supporters. Too many cases were observed which proved that his conclusions were incorrect.

Dixon strongly opposed the views of Gowers and became the outstanding champion of the route through the facial nerve. Dixon's impressions are summed up in the following quotation: "We must remember that we are not forced to accept these complicated courses (for taste innervation) for the geniculate ganglion is the homologue of the spinal ganglia just as is the gasserian of the fifth nerve. The nervus intermedius is considered to be continuous with the chorda tympani and so would represent a continuation of these fibers to the ganglion cells of the brain." He also emphasized the fact that in the human embryo, the chorda tympani is a direct branch of the facial nerve and in the early stages of development is not connected with the lingual nerve.

PATHWAY FOR TASTE THROUGH THE GLOSSOPHARYNGEAL NERVE

At least as early as 1834, the glossopharyngeal nerve was considered the nerve of taste for the entire tongue (Lussana). Biffi and Morganti (1846) determined the lingual supply to the anterior two thirds of the tongue and the glossopharyngeal supply to the posterior third. As previously indicated, Montault had already anticipated these observations. Despite the support favoring a fifth or seventh nerve supply for taste, there are those who believe that other evidence indicates that the ninth nerve supplies taste fibers to the entire tongue.

Eulenburg (1871), after obtaining negative results following section of the fifth and seventh nerve in animals, suggested that there must be a communication between the geniculate ganglion and the ninth nerve through the small superficial petrosal nerve, the tympanic plexus and Jacobson's nerve. Landois (1880) also taught that taste was carried by the ninth nerve, possibly by the route suggested by Eulenburg or by a branch passing directly from the facial nerve in the fallopian canal to the ganglion petrosum of the glossopharyngeal nerve.

Lehmann (1884) reported a cerebral injury with paralysis of the ninth nerve and none of the fifth; taste was reported lost over the entire one half of the tongue. Luciani (1915) in his textbook of physiology supported the glossopharyngeal theory. Recently Doyle (1923) cited a case in which taste was lost over one half of the tongue after periph-

eral avulsion of the ninth nerve, but the sensory root of the fifth had previously been avulsed. Although this case is cited to support the glossopharyngeal theory of taste, taste was not tested either before or after division of the trigeminus or before avulsion of the glossopharyngeal nerve. This evidence is therefore inconclusive as far as the anterior two thirds of the tongue is concerned.

It can readily be understood from the foregoing comment that tumors, inflammatory lesions, and cranial injuries have almost always affected more than one nerve and have therefore rendered any conclusions untrustworthy. The results of animal experimentation are so variable that no conclusions can be drawn.

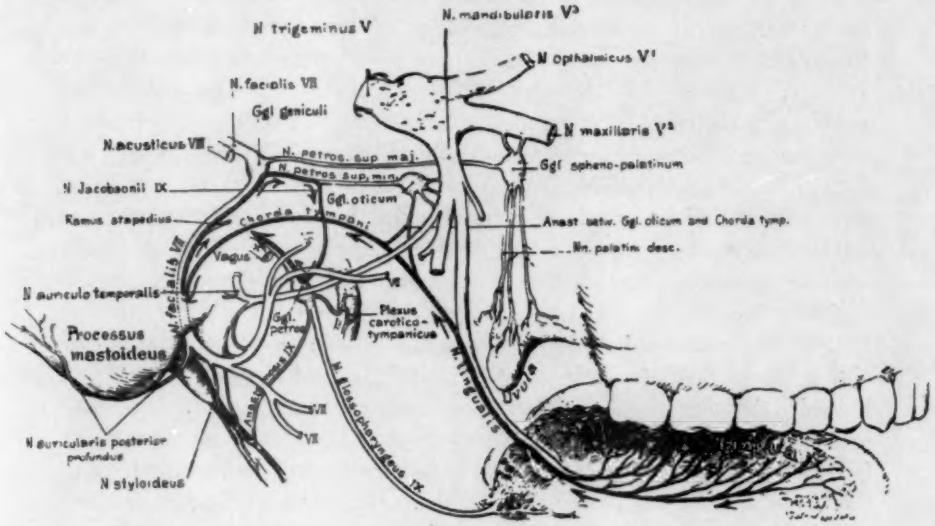

Fig. 4.—Pathway for taste fibers suggested by Eulenburg and Landois.

OPERATIONS ON PATIENTS

With the development of cranial surgery, a better opportunity was afforded to determine the effects on taste of section of the different cranial nerves. Krause (1895) divided intracranially the second division of the fifth nerve. Taste was not affected. Krause's observations did not support the views of Schiff and Erb, for taste was not affected when the great superficial petrosal and vidian nerves and the branches of the sphenopalatine ganglion were divided. Later the ganglion was removed from the same patient, and taste was lost. It seems natural, therefore, that Krause should have concluded that the third branch of the fifth conducted the fibers of taste.

Krause's work was quickly criticised by Dixon, who believed that the results were due to injury of the geniculate ganglion when the dura was stripped from the floor of the middle fossa. Dixon believed that both petrosal nerves might be stripped from their bed during exposure of the ganglion and that this ganglion might be pulled on or injured. Weakness of the facial muscles, even paralysis of these muscles in Krause's cases, seemed to support this contention.

Other surgeons began to report cases but with varying conclusions. Tiffany (1894) reported loss of taste after division of the second and third branches of the fifth nerve. Finney and Thomas reported loss of taste after extirpation of the ganglion in one case; in another, taste was preserved. Gowers cited the observations made on five patients operated on by Sir Victor Horsley and Ballance. Taste was lost in four and preserved in one. Horsley's results and views were much like those of Krause. Cushing in 1903 reported twenty cases of operation on the gasserian ganglion. In four, taste was impaired. Davies (1907) added twenty cases, the results almost duplicating those of Cushing. Taste was lost in one case, impaired in three and unchanged in sixteen. Krause later reported six cases, taste being lost in two, impaired in three and unchanged in one. Krause finally came to the conclusion, because of the variable observations, that taste fibers must pursue different courses in different individuals, and Oppenheim, Germany's leading neurologist at that time, concurred in this view. Harris (1926) published a series of eighty-six cases of tic douloureux in which alcohol had been injected into the third division of the fifth nerve or the gasserian ganglion. Taste was lost in eighty of these. Such an injection probably produces a more localized lesion than operations on the gasserian ganglion when approached in the usual way through the middle fossa. He was surprised to find, as were others before him, that although in a few cases taste was totally lost at first, it sometimes returned later, even when the tongue remained anesthetic. The objection can no longer be raised in these cases that the geniculate ganglion was injured in stripping the dura from the middle fossa. Harris, like Krause, expressed the belief that taste fibers have different courses in different persons, but that the fifth nerve is the principal nerve of taste. In explanation of these differences, he cited the work of Nageotte, in which it was shown that the fifth and sensory parts of the seventh and ninth nerves have a common nucleus which is continuous caudalward.

MATERIAL OF PRESENT STUDY

We have presumed to add to the lengthy literature on the course of taste fibers because we have material which has hitherto been unavailable. Pure isolated divisions of the fifth, seventh and ninth nerves have been

performed subtentorially for the relief of surgical conditions. Combined division of the fifth and seventh has also been performed. The fifth nerve has been divided intracranially at the pons for trigeminal neuralgia. The subtentorial operation has the advantage of not disturbing the petrosal nerves or geniculate ganglion or any other cranial nerve. Division of the seventh nerve alone has occurred during the removal of tumors of the cerebellopontine angle in a method recently described. As yet it has been impossible to extirpate these tumors without dividing the seventh nerve. As the fifth and ninth nerves are usually pushed aside by these tumors, they may not be injured when the tumor is removed. There is also an instance of a pure lesion of the seventh nerve which was accidentally injured when the auditory nerve was divided for relief from Ménière's disease. There was no disturbance of the fifth or ninth nerve in this case.

NECESSITY OF CONTROL OBSERVATIONS

Before presenting our evidence concerning the course of taste fibers derived from cases in which pure isolated lesions have been produced, it may be well to state something concerning (1) the methods of testing taste and (2) the necessity of making control tests. An appreciation of these two things may explain to some extent the divergent views held by different observers.

For a long time we were unable to understand why members of our own staff obtained different results. Three examiners would obtain results as variable as those reported in the literature. Even in a patient whose taste was found to be lost by one examiner, normal or subnormal taste would be found by another. Different results may be obtained for the following reasons: 1. Patients confuse touch and taste. 2. When patients are allowed to withdraw the tongue, taste will quickly be perceived by oral and posterior glossal taste buds. 3. At times, saliva coming in contact with the solution would carry the substance to be tested to the back of the mouth. 4. Patients are prone to guess, particularly when sensation is present. 5. By the sense of smell, patients may tell before tasting whether the solutions offered them are acid or bitter; solutions should therefore be used which do not stimulate smell.

In testing taste, the tip of the tongue should be covered with gauze and held by the examiner until the taste has been accurately identified in one of two ways: (1) The patient points to one of four tastes printed on a card, or (2) he nods when the examiner mentions the taste. The tongue is not to be drawn into the mouth until the taste has been definitely determined. If this method of examination is employed, the results will be accurate and uniform. Preoperative observations should be made as control experiments. Deviations from the normal occur

frequently and either unilaterally or bilaterally. Therefore unless taste is carefully tested before the operation, erroneous conclusions may be drawn. The acuity of taste varies considerably in different patients, as is indicated by the following observations.

CASES 1 and 2.—Two patients, both men, suffered with tic douloureux. Examination showed that neither had taste perception over the anterior two thirds of the tongue on either side when tested before operation.

CASE 3.—A man, aged 40, had a painful malignant ulcer of the tongue. He had no taste perception over the anterior two thirds of the tongue (either side) when tested before the operation.

CASE 4.—A man, aged 56, had had the gasserian ganglion removed seven years by the temporal route before the test was made. This patient had no sensation of taste on the anterior two thirds of the tongue on either side.

CASE 5.—A man, aged 65, entered the hospital for treatment for tic douloureux. When tested for taste perception before operation, it was found to be absent on the side of the neuralgia, but normal on the other side (anterior two-thirds). No cause for loss of taste on the affected side could be determined.

CASE 6 and 7.—A man, aged 49, and a woman, aged 62, were afflicted with tic douloureux. Before operation neither sugar nor salt could be detected on either side of the tongue (anterior two-thirds). Acid and bitter substances could be tasted, but perception was delayed.

CASE 8.—A woman, aged 45, was afflicted with tic douloureux. She could not taste sweet or bitter substances. She could taste sour substances after thirteen seconds and salt after twenty-three seconds. The observations on both sides were identical.

CASE 9.—A man, aged 60, with tic douloureux could not taste sugar, salt or bitter substances on either side of the tongue (anterior two-thirds). Sour substances could be tasted after eight seconds.

CASE 10.—A man, aged 63, afflicted with tic douloureux could not taste sugar or bitter substances. Perception of salt and sour were present but were delayed eight seconds.

It will be seen from the observations given that it cannot be assumed that taste is normal in any person. Each one should be tested before operation, for the incidence of defective taste is high and the character of the change is variable. Although loss or diminution in acuity of taste is usually symmetrical, this is not necessarily so, as is indicated by the hemigeusia in case 5. Had not taste been tested before operation in this case, section of the sensory root of the fifth nerve would have been regarded as the cause of the loss of taste.

There is still another possible cause of error. If alcoholic solutions of bitter substances are used diffusion occurs, and after a minute or more, taste may be perceived on the other side. The patient may or may not know on which side the taste perception occurs.

In all of our observations one minute has been the upper limit for taste determinations. Substances not detected within this time will not be appreciated at all.

PURE EXPERIMENTAL NERVE LESIONS

The material has been grouped under the following headings: (1) pure fifth nerve lesions, (2) pure ninth nerve lesions, (3) combined pure fifth and ninth nerve lesions, (4) pure seventh nerve lesions (intracranial) and (5) pure seventh nerve lesions (peripheral).

GROUP I: PURE FIFTH NERVE LESIONS

CASE 1.—A sparely nourished woman, aged 63, sought relief from trigeminal neuralgia. Pain like a pin prick began eleven years before examination, in the right side of the face below the eye. At first the attacks were not severe, but gradually they became more so and occurred more frequently; six years before, they became excruciating. They were induced by eating, drinking or touching the lip. Four injections of alcohol afforded relief over varying periods of time. The neurologic examination gave entirely negative results. Gustatory tests were not made before operation.

On Sept. 17, 1927, the right sensory root was divided by the subcerebellar route. Total anesthesia resulted for all forms of sensation in the domain of the trigeminal nerve.

Five months after operation, the results obtained by one of us (W. E. D.) in tests for taste were:

	Right (Affected Side), Seconds	Left Side
Salt	0	40 seconds
Acid	15	Instantly
Bitter	10	5 seconds
Sugar	0	0 seconds

CASE 2.—A rather obese woman, aged 70, sought relief for tic douloureux on the right side. Two years before consulting us she experienced sudden attacks of sharp pain, like needle pricks, in the right molar region. The pains became progressively more severe and more frequent. Gradually also the involved area of the face spread to the eye and the forehead. Eating, drinking, cold and touching the face precipitated the pain. The physical and neurologic examinations gave entirely negative results. Gustatory tests were not made before operation.

On March 30, 1927, the sensory root of the right trigeminal nerve was divided at the pons, by the subcerebellar route. Complete anesthesia for all forms of sensation followed in the domain of the trigeminal nerve.

Eight days after operation, the following results were obtained by one of us (W. E. D.) in a test for taste:

	Right (Affected Side), Seconds	Left
Sugar	15	Instantly
Salt	50	Instantly
Acid	20	Instantly
Bitter	60	Instantly

Eleven days after operation, the following results were obtained by another examiner:

	Right (Affected Side), Seconds	Left
Sugar	0	Instantly
Salt	0	Instantly
Acid	20	Instantly
Bitter	3	Instantly

Nine months after operation, the following results were obtained:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	0
Salt	0	0
Acid	2	2
Bitter	2	2

The results of a recent test, two and one-half years after operation were:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	30
Salt	2	2
Acid	2	1
Bitter	2	2

CASE 3.—A slender, emaciated woman, aged 51, was referred by Dr. John Gilmore, of Wheeling, W. Va., for relief from tic douloureux. Ten years ago a slight pain began in the lower teeth and the lower lip on the left side, without known cause. Gradually the pain became more severe and spread to the upper two branches of the nerve, thus involving the entire area of the right trigeminal nerve. The pain was paroxysmal and of terrific intensity. A draught of cold air, eating and drinking induced the attacks. All of the teeth on the left side had been extracted in a futile effort to abolish the pain. She had lost a great deal of weight because eating was painful. Physical and neurologic examinations yielded negative results. Section of the left sensory root of the trigeminal nerve at the pons (subcerebellar route) was performed on Feb. 7, 1928. Total anesthesia for all forms of sensation resulted in the domain of the affected nerve.

Before operation the following results were obtained by an associate in a test for taste on the affected side only:

	Left (Affected Side)
Sugar	7 seconds
Salt	15 seconds
Acid	8 seconds
Bitter	Not identified; called "sweetish"

Three days after operation, the following results were obtained by one of us (W. E. D.):

	Left (Affected Side), Seconds	Right, Seconds
Sugar	10	10
Salt	0	2
Acid	10	2
Bitter	10	2

Ten days after operation, the following results were obtained by an associate:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	5	5
Salt	20	10
Acid	18	10
Bitter	15	12

Case 4.—A large robust man, aged 42, was referred by Dr. John Gilmore, of Wheeling, W. Va., for treatment for tic douloureux. Six years before, tenderness was first noticed along the lower branch of the trigeminal nerve. Paroxysms of pain soon developed at the site of tenderness. The pain gradually increased in severity, and finally it involved all three branches of the nerve, though the starting point was in the mandibular branch. Never a day passed without several attacks of terrific pain. Eating, drinking and touching the face induced the pain. Physical and neurologic examinations gave entirely negative results.

On Feb. 7, 1928, the sensory root of the left trigeminal nerve was sectioned totally at the pons. Complete anesthesia over the domain of this nerve resulted.

Before operation the results of a gustatory examination on the affected side only, made by an associate, were as follows:

	Left (Affected Side)
Sugar	10 seconds
Salt	12 seconds
Acid	20 seconds
Bitter	15 seconds

Seven days after operation, the following results were obtained by one of us (W. E. D.):

	Left (Affected Side), Seconds	Right, Seconds
Sugar	30	20
Salt	Not recognized	5
Acid	2	2
Bitter	2	2

Ten days after operation, the following results were obtained by an associate:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	25	15
Salt	12	5
Acid	5	7
Bitter	20	10

Case 5.—A well nourished woman, aged 59, was referred by Dr. H. Klinzing, of Pittsburgh, for treatment for pain in the right side of the face. Fifteen years before she was seen by us the type of pain of which she complained began in the

right temple, soon passed to the eye, where it became more intense, and persisted for about twelve hours. There was some radiation of the pain to the upper and lower teeth on the same side, also to the back of the head and ear on the right side. The pain never spread to the left side, nor did it ever arise there. It was present almost every day and generally lasted about twelve hours. It usually began about 4 o'clock in the afternoon. Acetylsalicylic acid and a brand of amidopyrine relieved the patient only partially and for about two hours. Eating, drinking and rubbing the face did not induce the pain nor did anything else of which she knew. Physical and neurologic examinations yielded entirely negative results.

Because of the radiation of the pain to the teeth, a diagnosis of an unusual form of trigeminal neuralgia was made.

Complete section of the right sensory root at the pons was performed on July 28, 1927. Total anesthesia resulted for all forms of sensation in the affected nerve. Complete section of the sensory root was made because of the unusual type. There has not been a suggestion of the former pain to the time of this writing, two years later.

Before operation, the following results were obtained by an associate in examinations for taste:

	Right (Affected Side)	Left
Sugar	Instantly	Instantly
Salt	Instantly	Instantly
Acid	Instantly	Instantly
Bitter	Instantly	Instantly

Seven days after operation the following results were obtained by the same associate:

	Right (Affected Side), Seconds	Left
Sugar	2	2
Salt	2	Instantly
Acid	2	Instantly
Bitter	Instantly	Instantly

CASE 6.—A well nourished woman, aged 48, was referred by Dr. Louis Hamman. Her complaint was attacks of pain in the right eye. Since the age of 15, the patient had had occipital headaches extending to the vertex. About nine years before she consulted us this headache seemed to change to a single pain localized in the right eyeball. At first the pain occurred about every six weeks. Gradually it increased to once a week and became much more intense. It began gradually, at first being scarcely perceptible; it continued steadily to become more severe, reaching its maximum intensity in about twelve hours, and then gradually subsided. Each attack lasted about forty-eight hours. Codeine deadened the pain slightly. There was no pain in the face. Nothing induced the pain or modified the attack. At times there was a sensation of cold in the eyeball. The pain did not radiate to any part of the sensory domain of the trigeminal nerve. There was no known history of similar pain in any member of her family. The results of physical and neurologic examinations were normal.

The pain was suggestive of migraine. Relief from pain of seemingly similar character had been given by section of the sensory root in three other cases; this operation was performed on the right side on July 9, 1929. Total anesthesia resulted for all forms of sensation in the right terminal area.

Before operation, taste was found by an associate to be normal on both sides:

After operation (eight days), the following results were obtained by an associate:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	3	2
Salt	2	2
Acid	2	2
Bitter	2	2

Case 7.—A sparely nourished man, aged 58, was referred by Dr. Le Grand Guerry, of Columbia, S. C., because of bilateral tic douloureux. For twenty-one years he had suffered with trigeminal neuralgia of the right side; apparently all three branches were involved at the onset. The left side became involved six years before we saw him, but only the second and third branches were painful. The pains on both sides had all the characteristics of tic douloureaux. They were paroxysmal, and were brought on by eating, drinking, cold air or touching the face. He had had numerous peripheral operations and injections of alcohol, with short periods of temporary relief. Usually when the pain began on one side (either side) it spread to the other side, though there were periods when the attacks affected one side only.

The diagnosis was bilateral trigeminal neuralgia.

On April 30, 1926, both the right and left sensory roots of the trigeminal nerves were sectioned at the pons (subcerebellar route) at the same operation.

Tests for taste were not made before operation. Three weeks after operation, the patient could promptly taste sweet, salt, acid and bitter on both sides. The time of recognition of the taste was not noted, as this was our first test of this function. Six months later, he wrote: "The taste is perfect, so I can't understand why I am not gaining faster the sense of feeling or touch inside the mouth."

After the operation, both sides of the face were completely anesthetic to all forms of sensation.

Case 8.—A sparely nourished woman, aged 62, was referred for typical right-sided neuralgia of three years' duration. For over a year, the pains were only occasional and of very short duration; gradually, they became more frequent, of longer duration and more intense. Finally, each paroxysm of pain lasted about three minutes. They were induced by eating, drinking, cold air or touching the face. They began over the right eye and did not spread.

Physical and neurologic examinations gave entirely negative results.

The sensory root of the right trigeminal nerve was sectioned at the pons on Dec. 21, 1928. There was complete loss of all forms of sensation in the domain of the right trigeminal nerve after the operation.

Examination for taste was not made before or immediately after operation.

Seven months after the operation, the following results were obtained in tests for taste by one of us (W. E. D.):

	Right (Affected Side), Seconds	Left, Seconds
Sugar	1	4
Salt	1	40
Acid	1	40
Bitter	12	45

All forms of taste were much less intense on the left (normal) side.

TABLE 1 *(Group 1).—Results of Examinations for Taste, Over the Anterior Two Thirds of the Tongue, After Section of the Sensory Root of Trigeminal Nerve at the Pons; in Every Instance Total Anesthesia for all Forms of Sensation Has Followed in the Affected Trigeminal Domain; These Cases Are Therefore Cases of Pure Fifth Nerve Lesions*

Patient	Age	Diagnosis	Operation	Time of Test	Results of Sensory Examination Over Trigeminal Area	Time of Taste Perception		Control Tests (Before Operation)	
						Affected Side	Normal Side	Affected Side	Normal Side
F (Case 1)	63	Trigeminal neuralgia	Section of sensory root (cerebellar route)	5 months after operation	Total anesthesia to all forms of sensation	Sugar 0 Salt 0 Acid 15 seconds Bitter 10 seconds	0 40 seconds Instantly 5 seconds	No tests before operation	No tests before operation
F (Case 2)	70	Trigeminal neuralgia	Section of sensory root (cerebellar route)	8 days after operation	Total anesthesia to all forms of sensation	Sugar 15 seconds Salt 50 seconds Acid 20 seconds Bitter 60 seconds	Instantly Instantly Instantly Instantly	No tests before operation	No tests before operation
				11 days after operation		Sugar 0 Salt 0 Acid 20 seconds Bitter 3 seconds	Instantly Instantly Instantly Instantly		
				9 months after operation		Sugar 0 Salt 0 Acid 2 seconds Bitter 2 seconds	0 0 2 seconds 2 seconds		
				2½ years after operation		Sugar 0 Salt 2 seconds Acid 2 seconds Bitter 2 seconds	30 seconds 2 seconds 1 second 2 seconds		
F (Case 3)	51	Trigeminal neuralgia	Section of sensory root (cerebellar route)	3 days after operation		Sugar 10 seconds Salt 0 Acid 10 seconds Bitter 10 seconds	10 seconds 2 seconds 2 seconds 2 seconds	7 seconds 15 seconds 8 seconds 0	Not tested
				10 days after operation	Total anesthesia to all forms of sensation	Sugar 5 seconds Salt 20 seconds Acid 18 seconds Bitter 15 seconds	5 seconds 10 seconds 10 seconds 12 seconds		
M (Case 4)	42	Trigeminal neuralgia	Section of sensory root (cerebellar route)	7 days after operation	Total anesthesia to all forms of sensation	Sugar 30 seconds Salt 0 Acid 2 seconds Bitter 2 seconds	20 seconds 5 seconds 2 seconds 2 seconds	10 seconds 12 seconds 20 seconds 15 seconds	Not tested
				10 days after operation		Sugar 25 seconds Salt 12 seconds Acid 5 seconds Bitter 20 seconds	15 seconds 5 seconds 7 seconds 10 seconds		
F (Case 5)	50	Trigeminal neuralgia	Section of sensory root (cerebellar route)	7 days after operation	Total anesthesia to all forms of sensation	Sugar 2 seconds Salt 2 seconds Acid 2 seconds Bitter Instantly	2 seconds Instantly Instantly Instantly	Instantly Instantly Instantly Instantly	Instantly Instantly Instantly Instantly
F (Case 6)	48	Migraine-like pain in right eye	Section of sensory root (cerebellar route)	8 days after operation	Total anesthesia to all forms of sensation	Sugar 3 seconds Salt 2 seconds Acid 2 seconds Bitter 2 seconds	2 seconds 2 seconds 2 seconds 2 seconds	Recorded as normal	Recorded as normal
M (Case 7)	58	Bilateral trigeminal neuralgia	Section of both sensory roots (cerebellar route)	21 days after operation	Total anesthesia to all forms of sensation, on both sides	Sugar Promptly Salt recognized Acid Bitter	Promptly recognized	Not tested	
F (Case 8)	62	Trigeminal neuralgia	Section of sensory root (cerebellar route)	7 months after operation	Total anesthesia to all forms of sensation	Sugar 1 second Salt 1 second Acid 1 second Bitter 12 seconds	4 seconds 40 seconds 40 seconds 45 seconds	Not tested	

Summary.—In this group are included only those patients in whom total anesthesia for all forms of sensation except taste followed section of the sensory root of the trigeminus. There was essentially no difference in the acuity of taste after division of the sensory root. It may well be asked why these results should be different from those obtained by section of the sensory root by the temporal route as performed by Krause, Cushing and others. By the temporal route the petrosal nerves are torn and at times the geniculate ganglion is injured in stripping the dura from the temporal bone. Since the petrosal nerves have been included in the pathways of taste, their injury adds a complication and prevents the results from being accepted as pure fifth nerve lesions. By the subcerebellar route, which we have used exclusively, the lesion is strictly that of the fifth nerve, for no other nerves cross the line of approach.

Twenty-five additional cases of presumably total trigeminal section by this route are not included in this group because sensation in varying degrees has been retained. The preservation of sensation is not due, as might well be reasoned, to subtotal section of the sensory root but to variable adjacent accessory fibers which retain the sensory function. In none of these additional cases has taste been lost after partial or total section of the sensory root. Nor is a still greater number of cases of deliberate partial section of the sensory root included, although the results obtained are precisely similar.

Partial section of the nerve is now used exclusively instead of total division, for pain fibers have been found to be located solely in the posterior border of the sensory root and sensation to the face can be left almost intact by preservation of the anterior half of the root.

Perhaps the most impressive test of the series (group 1) that taste is not carried by the trigeminal nerve is offered in case 7, in which the sensory roots of both trigeminal nerves were totally divided at the same operation. Although all sensation of both trigeminal nerves was totally abolished, all forms of taste to the anterior two thirds of the tongue were promptly recognized.

GROUP II: PURE NINTH NERVE LESIONS

Case 1.—A robust man, aged 45, sought relief for agonizing, paroxysmal pains beginning in the tonsil region. These pains were brought on by swallowing and talking, and even occurred spontaneously. The pain was so terrific that he sat in terror, afraid to eat or drink, and held his head inclined at an angle so that the saliva could drool from his mouth on the unaffected side. These pains had been present for three years, though there were periods lasting for months in which the same stimuli would not produce the attacks. He said the pain felt as though a red hot poker were being thrust into the back of the tongue. He had learned that relief could be obtained by the application of cocaine to the affected jaw.

Physical and neurologic examinations gave entirely negative results. The history was typical of glossopharyngeal neuralgia.

On April 6, 1927, the glossopharyngeal nerve was divided intracranially under local anesthesia. Following the operation there was loss of sensation in the back of the tongue and the mouth on the affected side; this extended to the epiglottis below and to the roof of the pharynx above. There was also loss of taste in the posterior third of the tongue. The functions of the other cranial nerves remained entirely normal. A pure ninth nerve lesion was thus produced.

Test for taste by one of us (W. E. D.) on the anterior two thirds of the tongue, ten days after the operation, gave the following results:

	Left (Affected Side), Seconds	Right
Sugar	Instantly	Instantly
Salt	Instantly	Instantly
Acid	Instantly	Instantly
Bitter	Instantly	Instantly

Case 2.—A well nourished man, aged 56, pale and sallow from lack of recent nourishment, was referred by our associate, Dr. S. J. Crowe, for relief from pain that was characteristic of glossopharyngeal tic douloureux. Fifteen years before he was seen by us, while he was talking with a friend, there suddenly appeared an excruciating pain in a well localized spot in the back of the tongue and near the tonsils; it lasted only a few seconds. During the succeeding years, similar momentary pains struck him from time to time; they were so infrequent as not to be a severe handicap. The pain felt as though a red hot iron were being jabbed into the tongue. At first he was unable to discover any inciting cause, but later at certain times the attacks were brought on by eating, drinking and talking. For two weeks before entering the hospital the attacks had been almost continuous, and the patient did not dare to eat, drink or talk, sneezing or coughing also brought on the paroxysms.

Neurologic and physical examinations gave entirely negative results.

On May 11, 1927, under local anesthesia, the glossopharyngeal nerve was divided intracranially. There was loss of sensation over the same area as in the preceding case, and the same loss of taste in the posterior third of the tongue.

One week after the operation, the following results were obtained by one of us (W. E. D.):

	Left (Affected Side), Seconds	Right
Sugar	1	Instantly
Salt	1	Instantly
Acid	1	Instantly
Bitter	1	Instantly

At recent examination there was just an appreciable difference between the time of perception of taste, and to the patient there was also a slight difference in intensity of taste perception. Sensation over the domain of the fifth nerve was entirely normal.

Case 3.—A large, strong, colored man, aged 32, complained of a constant severe pain in the left side of the neck and ear. Four years before this examination a peculiar sensation developed in the left ear; at first it felt as if it were a bug and then a trigger zone in the external auditory canal. He was treated in the Johns Hopkins Dispensary for two years, at which time a swelling was found in the auditory canal.

During the first few months there was some discharge of pus, but later, although the swelling was incised on several occasions, it did not continue to drain

pus. The canal became occluded by a hard growth which was tender. From this, deafness and ringing in the ear developed. Two and one-half years ago a mastoid operation was performed, but no infection was found. A mass of fibrous and fatty tissue covered with epithelium was removed from the cartilaginous canal. Following this operation, hearing returned and there was relief from pain for over a year. One year ago, the present type of pain began; it gradually became worse so that he was unable to sleep at night without taking sedatives. The pain was continuous. It began in front of the ear, ran back of the ear and down the neck to the angle of the jaw. It was aggravated by swallowing cold air and by moving the jaw.

Physical and neurologic examinations showed nothing other than a localized lesion in the ear. The distribution of pain made us feel that the glossopharyngeal nerve was affected.

On Feb. 18, 1928, the left glossopharyngeal nerve was divided intracranially. No other nerves were injured. Before operation the following results were obtained by an associate in an examination for taste:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	10	10
Salt	12	10
Acid	5	7
Bitter	5	5

Eight days after the operation, the following results were obtained by the same associate:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	3	3
Salt	7	8
Acid	10	5
Bitter	4	3

Summary.—In these three cases of total section of the glossopharyngeal nerve, taste remained normal over the anterior two thirds of the tongue on the affected side. There can be no purer nerve lesions than those produced by intracranial section of the glossopharyngeal nerve, for no other nerves are touched during exposure and section of the nerve. The results of this group should, therefore, dispose of the ninth nerve as a conductor of taste from the anterior two thirds of the tongue.

GROUP III: COMBINED PURE FIFTH AND NINTH NERVE LESIONS

A large, somewhat undernourished man, aged 69, complained of severe facial pain caused by carcinoma of the left side of the face. The growth, which was of eleven years' duration, was first observed just within the left auditory canal. It had been treated with radium for the past eight years. It had healed and reopened on several occasions. The pain began two weeks after treatment with "radium seeds" at another clinic two years before. It had steadily become worse. At times the whole side of the face pained. Associated with the carcinoma there was necrosis of the neck of the mandible, from which the open sinus persisted.

TABLE 2 *(Group II.—Results of Examinations for Taste Over the Anterior Two Thirds of Tongue After Total Section of the Glossopharyngeal (IX) Nerve Intracranially*

Patient	Age	Diagnosis	Operation	Time of Test	Results of Sensory Examination Over Trigeminal (V) Area	Time of Taste Perception: Affected Side		Time of Taste Perception: Normal Side	Control Tests (Before Operation): Affected Side	Control Tests (Before Operation): Normal Side	Remarks
M (Case 1)	45	Glosso-pharyngeal neuralgia	Section of ninth nerve	10 days after operation	Normal	Sugar Salt Acid Bitter	Instantly Instantly Instantly Instantly	Instantly Instantly Instantly Instantly	Not tested	Not tested	Complete loss of taste over the posterior third of the tongue
M (Case 2)	56	Glosso pharyngeal neuralgia	Section of ninth nerve	7 days after operation	Normal	Sugar Salt Acid Bitter	1 second 1 second 1 second 1 second	Instantly Instantly Instantly Instantly	Not tested	Not tested	Complete loss of taste over the posterior third of the tongue
M (Case 3)	32	Glosso-pharyngeal neuralgia	Section of ninth nerve	8 days after operation	Normal	Sugar Salt Acid Bitter	3 seconds 7 seconds 10 seconds 4 seconds	3 seconds 8 seconds 5 seconds 3 seconds	10 seconds 12 seconds 5 seconds 5 seconds	10 seconds 10 seconds 7 seconds 5 seconds	Complete loss of taste over the posterior third of the tongue

TABLE 3 *(Group III).—Results in Examinations for Taste Over the Anterior Two Thirds of the Tongue After Total Section, Intracranially, of Both the Glossopharyngeal (IX) and the Trigeminal (V) Nerves*

Patient	Age	Diagnosis	Operation	Time of Test	Results of Sensory Examination Over Trigeminal (V) Area	Time of Taste Perception: Affected Side		Time of Taste Perception: Normal Side	Control Tests (Before Operation): Affected Side	Control Tests (Before Operation): Normal Side	Remarks
M (Case 1)	69	Carcinoma of the face and throat	Section of ninth and fifth nerves intracranially	7 days after operation	Complete anesthesia for all forms of sensation over the trigeminal domain	Sugar Salt Acid Bitter	30 seconds 35 seconds 10 seconds 20 seconds	Not tested	40 seconds 20 seconds 20 seconds 20 seconds	25 seconds 25 seconds 25 seconds 15 seconds	Complete loss of taste over the posterior third of the tongue

Section of the left fifth and ninth nerves intracranially and of the great auricular nerve superficially was performed on Feb. 21, 1928.

Before operation, the following results for taste were obtained by an associate:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	40	25
Salt	20	25
Acid	20	25
Bitter	20	15

Seven days after operation, the same associate obtained the following results for the anterior two thirds of the tongue on the affected side only:

	Left (Affected Side), Seconds
Sugar	30
Salt	35
Acid	10
Bitter	20

There was total anesthesia for all forms of sensation over the affected trigeminal area.

Summary.—This single instance of pure experimental lesions of the fifth and ninth nerves combined merely adds emphasis to the results of the two foregoing groups; namely, that the intracranial portion of neither the trigemnial nor the glossopharyngeal nerve conducts taste sensations from the anterior two thirds of the tongue.

GROUP IV: PURE SEVENTH NERVE LESIONS (INTRACRANIAL)

CASE 1.—A young woman, aged 25, of normal appearance, was referred with the diagnosis of an unlocalized tumor of the brain. Eight months before admission, headaches began. This was one month before her baby was born. Staggering occurred from time to time and made her fearful of crossing the street. Deafness began in the right ear and soon became total. During the last months of pregnancy, she had three attacks of projectile vomiting associated with severe headache. Two months after the birth of her baby, she consulted an ophthalmologist because of blurred vision; he found bilateral papilledema and advised her to consult a neurologist. Shortly afterward a cerebellar exploration was made in another clinic, but nothing was found. Five months later, symptoms having steadily progressed, she entered the Johns Hopkins Hospital.

The following positive observations indicated a right cerebellopontile tumor: (1) bilateral papilledema; (2) bulging, tight, bilateral cerebellar decompression; (3) deafness (total) in the right ear; (4) diminished sensation over the right trigeminal area; (5) slight right facial weakness of the peripheral type; (6) staggering gait; (7) a positive Romberg sign, falling to the right, and (8) ataxia on the right side.

In view of the negative cerebellar exploration at which the cerebellopontile angle had presumably been exposed, an injection of air was made. There was bilateral hydrocephalus, establishing the diagnosis of a subtentorial tumor.

On May 23, 1921, a right-sided cerebellopontile tumor was totally removed. The right facial nerve was necessarily sacrificed. A spinofacial anastomosis was refused.

Tests for taste were made on Dec. 20, 1928, or seven and one-half years after removal of the tumor. There had been no preoperative gustatory examination. All forms of sensation were normal over the domains of the trigeminal and glossopharyngeal nerves on the affected side.

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	5
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

Case 2.—A rather obese woman, aged 49, was referred by Dr. Hugh Morgan, of Nashville, Tenn., with the diagnosis of a cerebellopontile tumor. Twelve years before he consulted us, tinnitus began in the left ear; there was some associated dizziness. Six years before, deafness began in the left ear and steadily progressed. Three years before, unsteadiness of gait was first noticed. It was present only at times. There had been spells of unconsciousness with subsequent weakness of the lower limbs, but no convulsions. Headaches began in the suboccipital region four months before we saw him. There had also been left occipital pains and stiffness of the neck.

On neurologic examination, the patient showed the positive signs of a left cerebellopontile tumor; i. e., (1) complete deafness of the left ear; (2) a staggering gait; (3) a positive Romberg sign, falling backward and to the left; (4) ataxia on the left side, and (5) bilateral papilledema.

The diagnosis was a left cerebellopontile tumor.

On Nov. 3, 1923, the tumor was totally removed. The facial nerve was sacrificed. Spinofacial anastomosis gave excellent return of function.

Complete recovery followed. The patient was living and well on Aug. 1, 1929.

No tests for taste were made before operation. The sensation over the domain of the left (affected side) trigeminal and glossopharyngeal nerves was unimpaired.

In December, 1928, five years after the tumor had been removed, an examination for taste by one of us (W. E. D.) showed:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	0	5
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

Case 3.—A sparely nourished woman, aged 33, was referred by Dr. Louis Hamman, of Baltimore, with a diagnosis of tumor of the brain. Her symptoms began five years before we saw her with pain and stiffness in the back of the neck. These pains had grown worse and more frequent during the past two years, and they had been accompanied by dizziness and ringing in both ears. On three occasions she had lost consciousness. For about one year, she had had increasing dimness of vision and transient diplopia. There had also been violent vomiting every few days for the past six months. This was not associated with the intake of fluid but rather with a sudden change of position. For three months, she had been unable to walk without support. For the past year, she had been

much more irritable, unreasonable and irresponsible. For several months, there had been urgency and hesitancy in urination. Deafness was first noticed in the right ear two years before she consulted us.

Neurologic examinations disclosed a characteristic right cerebellopontile tumor, as revealed by the following symptoms: (1) total deafness of the right ear; (2) bilateral papilledema; (3) a staggering gait; (4) a positive Romberg sign; (5) ataxia on the right side, and (6) suggestive hyperesthesia over the right trigeminal area.

Operation: A right-sided cerebellopontile tumor was completely removed on Aug. 5, 1927. The facial nerve was sacrificed; this loss of function was repaired by a spinofacial anastomosis two weeks later.

A test for taste was made by one of us (W. E. D.) at the time of discharge from the hospital, eighteen days after operation, with the following results:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	10
Salt	0	15
Acid	0	7
Bitter	0	10

Sensation on the affected side of the face was unimpaired.

Case 4.—A well nourished woman, aged 29, was referred by Dr. John Barron, of York, S. C. She complained of staggering and drawing of her face. Five years before we saw her, deafness was first noticed in her left ear. About a year before, the deafness became more pronounced and other symptoms developed about the same time; there were headaches, attacks of dizziness and staggering, particularly when turning. Six months before examination, her gait became unsteady; she became worried and irritable and had frequent attacks of crying. The headaches became more frequent and more severe, especially during the menstrual periods. The headaches at first were frontal, and later became general. Vision was blurred and the patient had frequent attacks of diplopia. Four months before we saw her, numbness developed in the left side of the face, and at the same time her face drew to the right side and she was unable to close her left eye.

The following positive signs were disclosed on neurologic examination: (1) bilateral papilledema; (2) deafness in the right ear; (3) paralysis of the left side of the face; (4) a staggering gait; (5) a positive Romberg sign with falling toward the left; (6) nystagmus, and (7) bilateral ankle clonus and bilateral Babinski reflex.

On Sept. 13, 1927, a large left-sided cerebellopontile tumor was completely removed. The seventh nerve was sacrificed; two weeks later a spinofacial anastomosis was performed to correct the facial deformity.

Examination for taste made by one of us (W. E. D) at the time of discharge, seventeen days after operation, showed the following results:

	Left (Affected Side), Seconds	Right
Sugar	0	Instantly
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

Sensation on the affected side of the face was unimpaired.

Case 5.—The patient was a normal appearing woman, aged 42, referred by Dr. N. G. Wilson, of Norfolk, Va., with a diagnosis of tumor of the brain. The patient dated the onset of her present trouble to five years before we saw her, when she noticed a clicking, buzzing noise in the right ear. A month or two later she suddenly became deaf in that ear. She had very few symptoms, however, until a year before examination, when a tonsillectomy was done; immediately afterward she noticed numbness and loss of sensation in the right side of the face and the side of the cheek. At the same time pains which she considered sciatica appeared in both legs from time to time. For the past six months she had had staggering gait, with falling to the right. She had had some headache which concentrated in the right mastoid region and which was much intensified by straining at the stool.

The following positive neurologic observations indicated a right cerebellopontile tumor: (1) bilateral papilledema; (2) deafness in the right ear; (3) absence of corneal reflex on the right side, some loss of sensation in the right trigeminal area; (4) a positive Romberg sign, falling to the right; (5) a staggering gait, and (6) a positive Babinski reflex.

On May 5, 1925, a right-sided cerebellopontile tumor was completely removed. The right facial nerve was sacrificed; a spinofacial anastomosis was performed three weeks later. The patient has been well to the time of writing, Aug. 1, 1929.

Tests for taste were made on July 20, 1928, over three years after the tumor was removed. There had been no preoperative tests for taste. All forms of sensation over the domain of the trigeminal and glossopharyngeal nerves on the affected side were normal. The results of the tests for taste made by one of us (W. E. D.) were as follows:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	5
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

Case 6.—A slender woman, aged 35, was referred by Dr. Sydney Miller, of Baltimore, with the diagnosis of a tumor of the brain. Illness began two years before we saw her, with severe headaches in the frontal region on both sides. The headaches were usually associated with vomiting, which sometimes was suggestive of the projectile type. They had rather diminished in severity and frequency, though they were still very disturbing. Spells of dizziness began soon after the headaches; this persisted and was more marked when the patient moved about or turned the head suddenly. Impairment of hearing in the right ear was noticed about the same time; this had gradually progressed. The patient had felt weak in the knees. Her feet had seemed heavy. She had had the sensation of staggering.

Neurologic examination indicated a typical right-sided cerebellopontile tumor, as revealed by the following signs: (1) deafness of the right ear; (2) bilateral papilledema; (3) staggering gait; (4) a positive Romberg sign, with falling to the right; (5) ataxia on the right side; (6) nystagmus; (7) less active corneal reflex, and (8) diminution in sensation over the trigeminal area.

A right-sided cerebellopontile tumor was completely removed on Dec. 14, 1927. The seventh nerve was routinely sacrificed in the removal of the capsule. A spinofacial anastomosis was done two weeks later to correct this deformity.

A test for taste made by one of us (W. E. D) at the time of discharge, twenty-seven days after the operation, gave the following results:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	15
Salt	0	3
Acid	0	3
Bitter	0	5

Some diminution of all forms of sensation over the right trigeminal area remained.

CASE 7.—A well nourished man, aged 37, was referred by Dr. R. A. King, of Pittsburgh, with the diagnosis of a tumor of the brain. The symptoms began three years before our observation, with severe pains in the vertex. At the same time dizziness began to occur several times a day. He would stagger like a drunken man during these attacks. About two years before we saw him, weakness of the facial muscles on the right side was noticed. One year before, transient attacks of blindness appeared, lasting from a few seconds to a minute. Deafness antedated all of the symptoms mentioned; it began four years before examination and became complete two and one-half years ago.

Neurologic examination showed all the signs of a right cerebellopontile tumor, as follows: (1) bilateral papilledema; (2) weakness of the right external rectus muscle; (3) nystagmus; (4) absence of the corneal reflex; (5) diminished sensation over that side of the face; (6) partial right facial paralysis; (7) complete deafness in the right ear; (8) a staggering gait; (9) a positive Romberg sign, and (10) ataxia on the right side.

Operation: On May 5, 1927, a large cerebellopontile tumor on the right side was completely removed. Two weeks later a spinofacial anastomosis was done because of the facial paralysis resulting from the sacrifice of the seventh nerve.

At the time of discharge there still persisted a loss of the corneal reflex on this side and diminution of sensation over the right trigeminal area; this was less pronounced than before operation.

The following results were obtained in a test for taste made by one of us (W. E. D.) at the time of discharge, three weeks after operation:

	Right (Affected Side), Seconds	Left
Sugar	0	Instantly
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

CASE 8.—A feeble, emaciated woman, aged 58, was referred by Dr. A. E. Fink, of Newark, N. J., with the diagnosis of a cerebellopontile tumor. Three years before, she had influenza, followed by otitis media on the left side. The hearing in the left ear had since been impaired. For a year she had been totally deaf in the left ear and for the past six months she had noticed an instability of balance, with a tendency to fall to the left when the eyes were closed; she had, however, never fallen. In the left ear there had been a noise like an engine.

Neurologic examination showed the following positive observations: (1) complete deafness in the left ear; (2) slight facial weakness on the left side; (3) diminished corneal reflex and impaired sensation on the left side; (4) a positive

Romberg sign and staggering gait; (5) nystagmus, and (6) ataxia on the left side.

On April 24, 1926, a left cerebellopontile tumor was completely removed. The left facial nerve was sacrificed in removing the tumor.

An examination for taste, made by one of us (W. E. D.) two and one-half years after the operation, gave the following results:

	Left (Affected Side), Seconds	Right
Sugar	0	Instantly
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

There was a slightly diminished acuity for heat, cold and sharp over the left trigeminal area. There was no loss of sensation over the glossopharyngeal domain.

Case 9.—A normal appearing woman, aged 28, entered the Johns Hopkins Hospital to be treated for a tumor of the brain. Her first symptoms began two years before with beginning deafness in the right ear. Six months before admission to the hospital she became totally deaf in this ear, and the hearing on the left side had begun to diminish. For the past six months she had had a staggering gait. Three months before examination this became so severe that she fell on several occasions, always to the right side. At the same time her speech became thick and slurred, and difficulty in swallowing developed. Recently her right arm and right leg had been useless. She had had severe general headaches with intensification in the supra-orbital region, and more on the right side.

Neurologic examination indicated a right cerebellopontile tumor, as revealed by the following signs: (1) bilateral papilledema; (2) deafness in the right ear; (3) some diminution in sensation in the left side of the face; (4) slight facial weakness on the right side, peripheral in type; (5) staggering gait; (6) a positive Romberg sign, falling backward and to the right; (7) nystagmus; (8) ataxia and adiadokokinesia, and (9) bilateral Babinski reflex and bilateral ankle clonus.

A right cerebellopontile tumor was completely removed on July 16, 1926. The facial nerve was sacrificed. Fourteen months later an examination for taste made by one of us (W. E. D.) showed:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	3
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

Sensory examination of the fifth nerve showed slightly diminished acuity for pin prick. Sensation for heat and cold was normal and equal on the two sides. There was no evidence of disturbance of the glossopharyngeal nerve.

Summary.—This group offers the positive side of the taste problem. In every instance all forms of taste were immediately and permanently abolished when the facial nerve was divided intracranially. Taste therefore is conveyed to the brain stem from the geniculate ganglion by the intracranial portion of the facial nerve (the nerve of Wrisberg). So far as is known, these are the only pure experimental lesions of the intracranial portion of the facial nerve.

In the last four cases of this group the lesions were not pure, for some impairment of trigeminal sensation persisted. This was due to stripping the tumor from the sensory root of the trigeminus. The results, however, were identical with those for the pure lesions of the nerve as represented by the first five cases of the group.

GROUP V: PURE SEVENTH NERVE LESIONS (PERIPHERAL)

CASE 1.—A young girl, aged 17, was operated on for mastoid at another clinic eighteen months before we saw her. Since the operation there had been complete facial paralysis on the corresponding side. No other cranial nerves were affected. Sensation over the trigeminal and glossopharyngeal areas was normal.

A test for taste, made by one of us (W. E. D.) eighteen months after section of the facial nerve, gave the following results:

	Right (Affected Side), Seconds	Left
Sugar	0	Instantly
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

CASE 2.—A well nourished man, aged 24, was operated on for chronic mastoid infection at another hospital ten months before we saw him. Since the operation the facial nerve has been totally paralyzed. There has been no loss of function in the trigeminal or glossopharyngeal areas. Hearing is still intact.

An examination for taste made by one of us (W. E. D.) gave the following results:

	Left (Affected Side), Seconds	Right
Sugar	0	Instantly
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

CASE 3.—In a woman, aged 30, ten days before she came under our observation there was sudden complete paralysis of the facial muscles on the left side during the night. There had been a prodrome of pain in the mastoid region for thirty-six hours before paralysis developed. No other cranial nerves were affected. Hearing was normal. The trigeminal and glossopharyngeal sensory domains were normal.

An examination for taste made by one of us (W. E. D.) ten days after the paralysis gave the following results:

	Left (Affected Side), Seconds	Right
Sugar	0	Instantly
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

CASE 4.—A man, aged 54, had an injury to the head fifteen months before we saw him. He was unconscious for twelve hours, after which consciousness gradually returned. There was no weakness of either arm or leg after the accident, and no disturbance of speech; his hearing was normal in both ears. Sensation over

TABLE 4 *(Group IV).—Results of Examinations for Taste Over the Anterior Two Thirds of the Tongue After Total Section of the Facial (VII) Nerve Intracranially (in Removal of Cerebellopontine Tumor); Sensation Over the Trigeminal Domain Was Subsequently Intact in Five Cases, and Somewhat Impaired in Four Cases*

Patient	Age	Diagnosis	Operation	Time of Test	Results of Sensory Examination Over Trigeminal (V) Area	Time of Taste Perception		Control Tests (Before Operation)	
						Affected Side	Normal Side	Affected Side	Normal Side
F (Case 1)	25	Cerebello-pontine tumor	Total removal of tumor	7½ years after extirpation of tumor	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	5 seconds Instantly Instantly Instantly	Not tested	Not tested
F (Case 2)	49	Cerebello-pontine tumor	Total removal of tumor	5 years after extirpation of tumor	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	5 seconds Instantly Instantly Instantly	Not tested	Not tested
F (Case 3)	33	Cerebello-pontine tumor	Total removal of tumor	18 days after extirpation of tumor	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	10 seconds 15 seconds 7 seconds 10 seconds	Not tested	Not tested
F (Case 4)	29	Cerebello-pontine tumor	Total removal of tumor	17 days after extirpation of tumor	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly Instantly Instantly Instantly	Not tested	Not tested
F (Case 5)	42	Cerebello-pontine tumor	Total removal of tumor	3 years after extirpation of tumor	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	5 seconds Instantly Instantly Instantly	Not tested	Not tested
F (Case 6)	35	Cerebello-pontine tumor	Total removal of tumor	27 days after extirpation of tumor	Some diminution of all forms of sensation	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	15 seconds 3 seconds 3 seconds 3 seconds	Not tested	Not tested
M (Case 7)	37	Cerebello-pontine tumor	Total removal of tumor	21 days after extirpation of tumor	Some diminution of all forms of sensation	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly Instantly Instantly Instantly	Not tested	Not tested
F (Case 8)	58	Cerebello-pontine tumor	Total removal of tumor	2½ years after extirpation of tumor	Slightly diminished perception of heat, cold and sharp	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly Instantly Instantly Instantly	Not tested	Not tested
F (Case 9)	28	Cerebello-pontine tumor	Total removal of tumor	14 months after extirpation of tumor	Perception of sharp diminished; sensation otherwise unaffected	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	3 seconds Instantly Instantly Instantly	Not tested	Not tested

the trigeminal and glossopharyngeal areas was normal. There had been complete paralysis of the left side of the face since the accident.

An examination for taste, made by one of us fifteen months after the accident, gave the following results:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	0	20
Salt	0	0
Acid	0	5
Bitter	0	30

CASE 5.—A young girl, aged 15, was operated on at another clinic for chronic mastoid infection of six years' duration. Following the operation, twelve months before we saw her, she had complete paralysis of the right side of the face. No other cranial nerves were affected.

A test for taste made by one of us (W. E. D.) at this time gave the following results:

	Right (Affected Side), Seconds	Left
Sugar	0	Instantly
Salt	0	Instantly
Acid	0	Instantly
Bitter	0	Instantly

CASE 6.—A youth, aged 18, was operated on at another clinic for a chronic mastoid infection of ten years' duration. The operation was performed seven months before the patient came under our observation.

Tests for taste made by one of us (W. E. D.) at this time gave the following results:

	Right (Affected Side), Seconds	Left, Seconds
Sugar	0	5
Salt	0	20
Acid	0	3
Bitter	0	7

CASE 7.—A youth, aged 16, was operated on at another clinic for chronic mastoid infection of two years' duration. An operation was performed three months before we saw the patient.

A test for taste made by one of us (W. E. D.) at that time gave the following results:

	Right (Affected Side), Seconds	Left
Sugar	0	Instantly
Salt	0	5 seconds
Acid	0	Instantly
Bitter	0	Instantly

CASE 8.—A large, robust, well nourished man, aged 47, was referred by Dr. F. C. Schreiber, of Washington, D. C., for treatment for Ménière's disease. One year before examination, when he awoke and raised his head to get out of bed he felt dizzy and vomited, but had no nausea. For three days this dizziness

persisted and prevented him from getting out of bed. Every time he moved, the dizziness was intensified and frequently caused vomiting. After the third day he was able to get out of bed, but still remained more or less dizzy for three weeks, at which time the attack had entirely cleared. Four months later he had another attack, almost exactly similar to the previous one. This attack lasted one week. Six months after the first attack, partial deafness appeared in the left ear and, at the same time, tinnitus; both the deafness and the tinnitus came on suddenly during an attack of dizziness.

The neurologic examination showed partial deafness of the left ear, staggering gait and a positive Babinski reflex on the left side. The diagnosis was Ménière's disease.

On April 12, 1927, section of the eighth nerve, left, was performed. The facial nerve was accidentally injured, producing complete facial paralysis, for which a spinofacial anastomosis was done later.

The results of an examination for taste made by one of us (W. E. D.) after the operation were as follows:

	Left (Affected Side), Seconds	Right, Seconds
Sugar	0	0
Salt	0	5
Acid	0	5
Bitter	0	10

Summary.—The results of these cases will clear any doubt which may yet remain that the facial nerve conducts sensations of taste from the point of union with the chorda tympani nerve with the geniculate ganglion. The results shown in group 4 indicate that the sensations of taste are conducted by the chorda tympani through the nervus intermedias, which represents the sensory portion of the seventh nerve of the gustatory nucleus in the pons.

SUMMARY OF RESULTS IN THE ENTIRE SERIES OF EXPERIMENTS

In this series of experiments on man, made after intracranial division of the different nerves presumably concerned with taste, several facts have been determined. Intracranial division at the brain stem of the glossopharyngeal nerve or of the sensory root of the fifth nerve or of both nerves is not accompanied by permanent loss of taste to the anterior two thirds of the tongue. Total loss of taste on the anterior two thirds of the tongue invariably follows intracranial division of the seventh nerve (at the pons) or of the peripheral portion at any point between its exit from the pons and the geniculate ganglion, from which point the fibers pursue a separate course.

At times there may be a temporary loss or diminution in acuity of taste following operations on the gasserian ganglion or the sensory root of the fifth nerve, but later taste returns to normal. Initial loss of taste, with subsequent return, has been noted by Köster and Cushing after operations on the gasserian ganglion. They assumed that another

TABLE 5 *(Group V).—Results of Examinations for Taste, Following Complete (Peripheral) Facial (VII) Paralysis, Resulting from Various Causes, Principally Mastoid Operations; Sensation Over the Trigeminal Area Is Normal in Every Case*

Patient	Age	Diagnosis	Operation	Time of Test	Sensory Examination	Time of Taste Perception		Control Tests (Before Operation)	
						Affected Side	Normal Side	Affected Side	Normal Side
F (Case 1)	17	Facial paralysis	Mastoid	18 months after injury to nerve	Normal	Sugar. ..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly Instantly Instantly Instantly	Not tested	Not tested
M (Case 2)	24	Facial paralysis	Mastoid	10 months after facial paralysis	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly Instantly Instantly Instantly	Not tested	Not tested
F (Case 3)	30	Bell's palsy	None	10 days after paralysis	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly Instantly Instantly Instantly	Not tested	Not tested
M (Case 4)	54	Fractured skull	None	15 months after paralysis	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	20 seconds 0 5 seconds 30 seconds	Not tested	Not tested
F (Case 5)	15	Facial paralysis	Mastoid	12 months after paralysis	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly Instantly Instantly Instantly	Not tested	Not tested
M (Case 6)	18	Facial paralysis	Mastoid	7 months after paralysis	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	5 seconds 20 seconds 3 seconds 7 seconds	Not tested	Not tested
M (Case 7)	16	Facial paralysis	Mastoid	2 years after paralysis	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	Instantly 5 seconds Instantly Instantly	Not tested	Not tested
M (Case 8)	47	Ménière's disease	Section of auditory nerve	3 weeks after injury of facial nerve	Normal	Sugar..... 0 Salt..... 0 Acid..... 0 Bitter..... 0	0 5 seconds 5 seconds 10 seconds	Not tested	Not tested

nerve had taken over the function of conducting sensations of taste in these cases. It seems improbable in the present cases that another nerve will assume the function of conducting the sensation of taste. We have no experimental or clinical observations which will warrant such an assumption.

STUDIES ON A PATIENT WITH MULTIPLE, SMALL METASTATIC TUMORS OF THE CRANIAL NERVES

Despite the untrustworthiness of the gustatory changes produced by primary or secondary tumors involving nerves conducting sensations of taste, the case about to be recorded is different from those which have been reported. It is included in this paper only because the changes in taste were so closely and definitely associated with loss of function of the seventh nerve occurring first on one and then on the other side. At autopsy many tiny, discrete, secondary melanotic sarcomas were found in and along the nerves of the cauda equina, and a few nodules in the cranial nerves on both sides (fig. 5).

Each nodule was so small that the tumor could cause only local destruction of the nerve involved, and could not press on adjacent cranial nerves. Moreover, the loss of taste occurred during the stay of the patient in the hospital and coincided exactly with the loss of function of the facial nerves.

A young man, aged 25, was under observation in the hospital over a period of three months. When first seen, his main complaint was referable to the cauda equina, but a peripheral facial paralysis was also noted on one side. In the early stages, the right facial paralysis was the only sign of intracranial disturbance. Taste was entirely lost over the anterior two thirds of the tongue on the right side. It was unaffected on the left side. Sensation, including the corneal reflex, was normal over the area of distribution of both fifth nerves. The action of the masseter, temporal and pterygoid muscles was unaffected. After a few weeks, paralysis of the left facial muscles began to develop. Within ten days this became complete, the typical mask appearance of bilateral facial paralysis developing. Coincidently, taste was lost on the left side. Loss of taste was noted over the anterior two thirds of the tongue on both sides, though neither trigeminal nerve was as yet affected. Some time later, some involvement of the right trigeminus was noted. Although this gradually progressed, it never became complete. The left trigeminus was unaffected. Toward the end, the auditory, glossopharyngeal and vagus nerves were gradually affected. Total deafness finally ensued. Dysphagia was soon followed by total inability to swallow.

CONCLUSIONS

We find no evidence to support the theory that a variable nerve supply conducts the sensations of taste, as first advanced by Krause and more recently sponsored by Harris who accepted Nageotte's anatomic demonstration that the fifth, seventh and ninth nerves have a common gustatory nucleus in the pons.

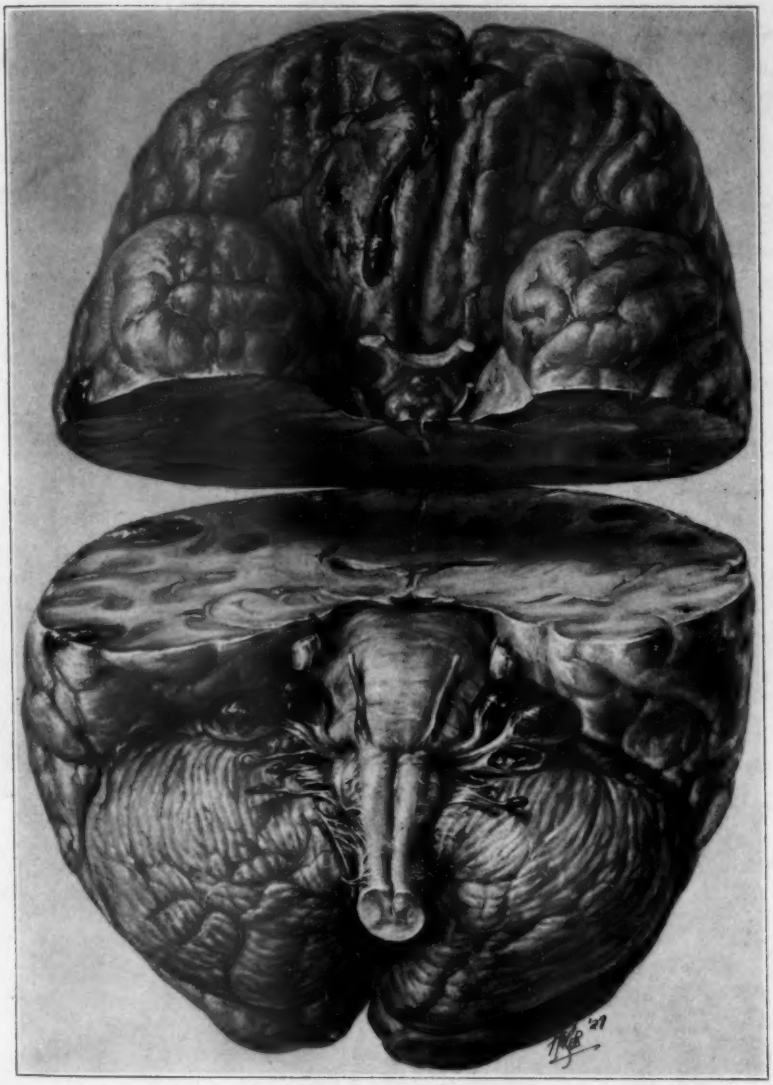

Fig. 5.—Drawing of brain showing numerous metastatic nodules in the cranial nerves. The importance of this specimen in connection with the pathway of taste lies in the fact that the nodules destroyed both seventh nerves and both fifth nerves were unaffected except for a tiny nodule in the right motor root. Taste was lost on both sides (anterior two thirds of the tongue) while sensation over the sensory domain of the fifth nerves remained intact.

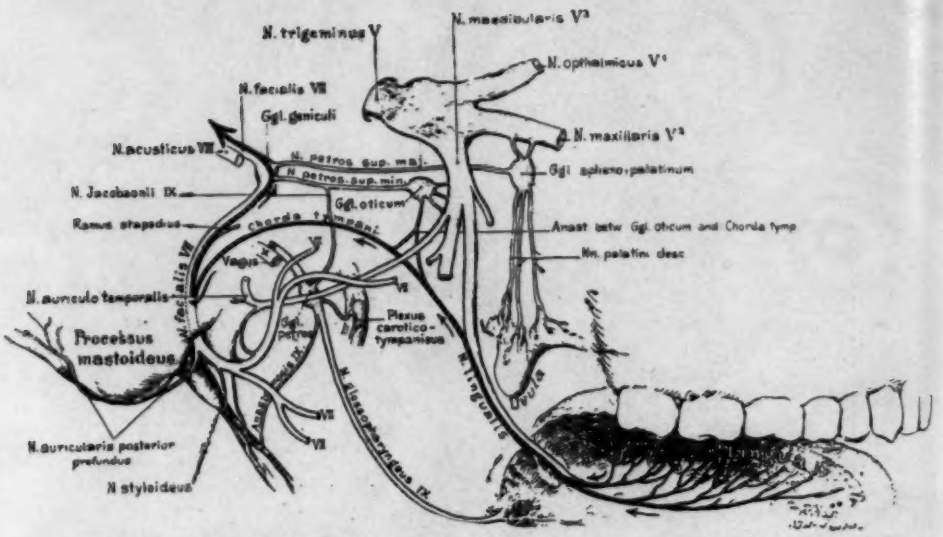

Fig. 6.—Pathway of taste from the anterior two thirds of the tongue as determined by our observations after intracranial division of the fifth and seventh nerves (Lewis and Dandy). This pathway was first suggested by Lussana.

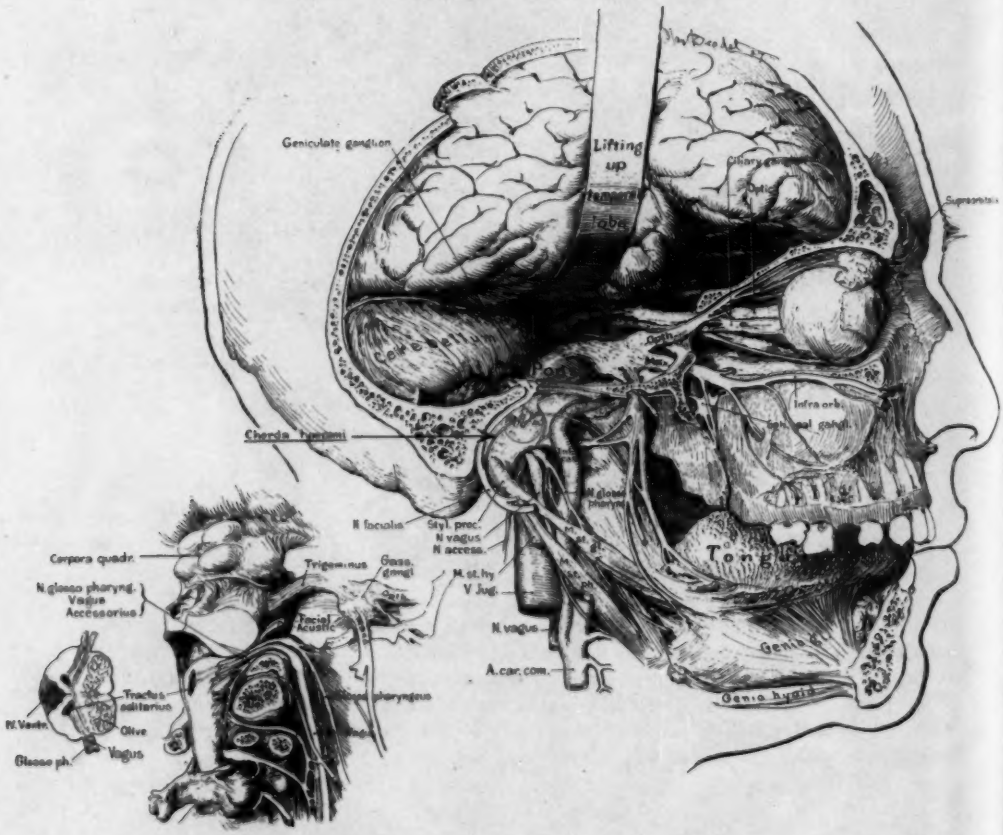

Fig. 7.—Drawing by Mr. Brödel to show the course of the taste fibers which we believe to be from the tongue through the chorda tympani to the facial nerve, thence directly through the geniculate ganglion and nerve of Wrisberg to the tractus solitarius in the pons.

Our observations indicate that but one nerve conducts sensations of taste from the anterior two thirds of the tongue. This is the nervus intermedius (Wrisberg) or the glossopalatinus (Hardesty), which represents the sensory portion of the seventh nerve.

The course of the pathway of taste is direct. Stimuli pass from the taste buds through the chorda tympani by way of the geniculate ganglion and the nervus intermedius into the pons.

These conclusions are based on the results of intracranial division of the isolated cranial nerves in patients.

The following are the observations on which the conclusions are drawn: 1. Section of the sensory root of the fifth nerve is not followed by any permanent changes in taste, although in each instance all forms of sensation except taste over the trigeminal domain are totally absent. 2. After intracranial division of the seventh nerve, including the sensory portion (nervus intermedius of Wrisberg, the N. glossopalatinus of Hardesty), taste to the anterior two thirds of the tongue is completely and permanently lost. 3. Division of the facial nerve, between the geniculate ganglion and the point at which the chorda tympani leaves the facial nerve to pass through the middle ear, is invariably followed by total and permanent loss of taste. 4. Intracranial division of the glossopharyngeal nerve is followed by complete and permanent loss of taste to the posterior one third of the tongue, but there is no effect on taste in the anterior two thirds of the tongue.

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PAINFUL POSTOPERATIVE ABDOMINAL SCARS*

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A considerable number of patients complain of postoperative soreness in the region of their scars, often of enough severity to interfere with the pursuance of their daily routine. Some of these conditions are often diagnosed as postoperative adhesions. Frequently, at secondary exploration, insufficient pathologic change is found to account for the symptomatology. A diagnosis of neurasthenia may be made as a result. It is my opinion that a certain number of these cases may be due to neuromas in the abdominal scar.

The literature in regard to neuromas or neuritis of the abdominal scars is scant, and it seems of sufficient import to review what literature there is and to report four cases which tend to show that this condition is more prevalent than is generally believed.

The microscopic proof of this lesion is extremely difficult, as the nerves traversing the scar tissue are extremely small and their identification in the excised tissue almost impossible.

The assumption that the symptoms and pathologic changes in the four cases mentioned subsequently were due to neuroma or traumatic neuritis of the lower thoracic nerves is based on the following data: 1. In three cases the injection of procaine hydrochloride into the fascia of the right rectus muscle relieved the pain for various periods of time. 2. The patients in three cases were relieved from symptoms by excision of the scar tissue en bloc without opening the peritoneum. In the fourth case the peritoneum was opened to inspect the abdominal viscera, and there was insufficient pathologic change in the peritoneal cavity to account for the patient's symptoms. 3. The peripheral pain in amputation stumps has been definitely shown to be due to neuroma, and the microscope has confirmed this diagnosis.

As the process of repair, with contraction of scar tissue and injury to nerves, is similar in amputation stumps and in abdominal scars, it is reasonable to assume that postoperative pain in the abdominal wound may therefore be due to neuroma.

In order to refresh the memory, an account of the anatomy of the lower six thoracic nerves is given (fig. 1).

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ANATOMY OF THE LOWER SIX THORACIC NERVES

The seventh, eighth, ninth, tenth and eleventh thoracic nerves differ from the upper thoracic nerves only in regard to a part of their course and distribution. Each has the same course and communications as the preceding nerves in the thoracic wall. In addition, these nerves have a further course and distribution in the abdominal wall. Each nerve traverses its intercostal space in the way described. At the anterior

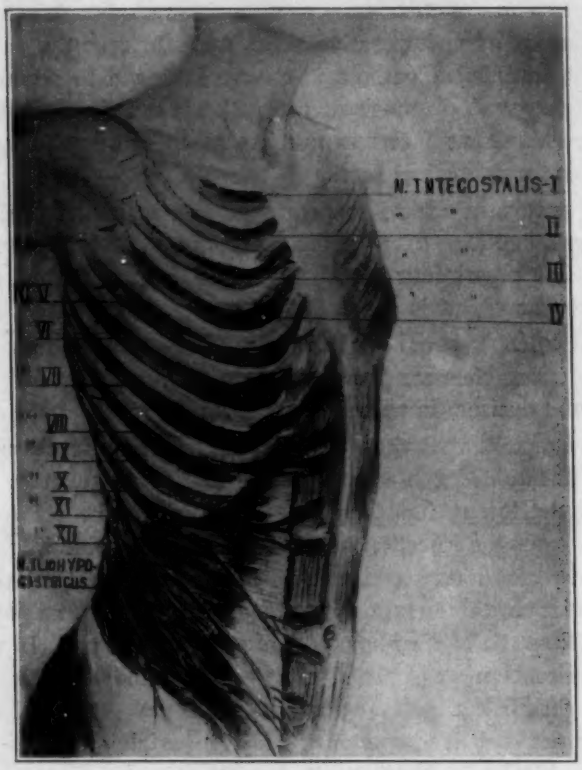

Fig. 1.—Diagrammatic sketch showing abdominal distribution of the lower six intercostal nerves. Note that the external oblique and the internal oblique muscles have been removed.

end of the space, the nerve pierces the attachment of the diaphragm and the transversalis abdominis muscles to the costal cartilages, and courses forward in the abdominal wall between the transversalis and obliquus internus muscles. The nerve then passes between the rectus muscle and the posterior layer of its sheath, and eventually reaches the anterior abdominal wall and becomes cutaneous by the piercing of the rectus itself and the anterior layer of its sheath.

Muscular Branches.—The lower intercostal nerves supply the intercostal muscles of the spaces in which they lie; and in the abdominal wall they innervate the transversalis, obliqui and rectus abdominis. The branches arise from the main trunk as well as from the lateral and anterior branches.

Cutaneous Branches.—The cutaneous branches are lateral and anterior. The lateral branches divide into anterior and posterior parts, and, becoming superficial along the line of interdigitation of the obliquus externus muscle with the serratus magnus and latissimus dorsi, they are directed more obliquely downward than the lateral branches of the higher intercostal nerves, and are distributed to the skin of the loin as low down as the buttock. The lateral branch of the eleventh nerve can be traced over the iliac crest.

The anterior branches are small. That of the seventh nerve innervates the skin at the level of the ensiform cartilage. The eighth and ninth appear between the ensiform cartilage and the umbilicus; the tenth nerve supplies the region of the umbilicus, and the eleventh the area immediately below the umbilicus.

The cutaneous branches of these nerves, including the posterior primary divisions, thus supply continuous belts of skin, which can be mapped out on the body from the vertebral column behind to the middle line in front. These nerves are not placed horizontally but tend to be drawn downward as the series is followed from the upper to the lower nerves.

The twelfth thoracic nerve is peculiar in its course and distribution. It emerges below the last rib and passes outward and downward in the posterior abdominal wall under cover of the psoas muscle, and between the external arcuate ligament and the quadratus lumborum muscle; it pierces the transversalis muscle, and courses forward in the interval between it and the obliquus internus as far as the sheath of the rectus muscle. After piercing the posterior layer of the sheath, the rectus muscle and the anterior layer of the sheath, it terminates by supplying the skin of the anterior abdominal wall midway between the umbilicus and the pubis. The branches of the nerve are the muscular, to the transversalis, obliqui, rectus and pyramidalis muscles of the abdominal wall, and the cutaneous, two in number—an anterior terminal branch, which supplies the skin of the anterior abdominal wall midway between the umbilicus and the pubis, and a large lateral cutaneous (iliac) branch, which, passing obliquely downward through the lateral muscles of the abdominal wall, becomes superficial above the iliac crest about 2 inches behind the anterior superior spine. It supplies the skin of the buttock as far down as a point below and in front of the great trochanter of the femur.

The twelfth thoracic nerve, in many cases, receives a communicating branch from the eleventh, near its origin, and still more frequently sends a fine branch to join the origin of the first lumbar nerve in the psoas muscle. It may communicate also with the iliohypergastric nerve, as they lie together in the abdominal wall.

When a nerve is cut completely or in part, the neuraxes start to grow in a straight line, if their growth is not interrupted. At the same time, however, there is a contraction of the scar tissue in the periphery of the scar, so that frequently thwarted neuraxes try to force their way through the scar tissue and twist themselves into spirals and corkscrew-like bodies. They are called perroncito fibrils.

Jager and Traum studied this process of nerve regeneration in man in about fifty operative scars, mostly laparotomy scars, using more than 1,000 histologic sections. The process of nerve regeneration, as found in these studies, was as follows:

> Degeneration resulted often from section of the nerve fibers at operation, and degenerative changes are evident in the nerve fibers in the first few weeks postoperative. In a four weeks old scar, however, the products of nerve degeneration had disappeared. Nerve elements in a scar of this age were not demonstrable in the stratum papillare or reticulare as a rule; but in the tela subcutanae there were thin nerve fibers, usually grouped in bundles, extending to the epithelium. Other fibers followed the course of the arterioles and capillaries in the scar tissue. In older scars the nerve fibers advanced into the epithelium, and in scars ten or twelve weeks old had reached the stratum papillare. As they advanced the fibers tended more and more to show dichotomous division with the formation of collateral processes, which often anastomosed with neighboring fibers. Most of the nerve fibers in the scar tissue showed no medullary substance. No tendency to form encapsulated end corpuscles was observed in scar tissue. The nerve endings were primitive in type.

These authors, however, did not study the nerve repair in the deeper layers of the scar.

Quain and Eggers, in a review of painful scars observed during a routine examination of recruits for the army, found that pain in and about an abdominal scar, often deep seated or radiating to the back, was observed in a certain percentage of cases in a military hospital. They described several types of cases in which operations revealed either hernia or adhesions beneath the wound. They also stated that there is another type of painful scar which they were not able to demonstrate clearly in this series of cases—cases in which nerve fibers are caught in the scar and produce pain. To prove this condition, a local anesthesia can be injected into the abdominal zone containing the nerves leading to a scar and in the positive cases all pain will cease. Excision of the scar will bring about a cure.

In 1924, Marshall Clinton read a paper before the Section on Surgery of the American Medical Association, entitled "Subcostal

Neuritis as a Cause of Abdominal Pain." Clinton claimed that the lower intercostal nerves, particularly the eleventh and twelfth, as they pass beyond the tips of the last few ribs, are frequently susceptible to trauma. He claimed that the commonest cause of injury of the eleventh and twelfth nerves is by repeated squeezing of the nerve between the tips of the rib and the wing of the pelvis. This particular trauma is found in short-waisted persons or in those who have an unusually long eleventh or twelfth rib. The pain is characterized by being of a tooth-ache type, accentuated in the morning when the patient arises and bends over. The pressure under the tip of the eleventh or twelfth rib will reproduce this pain. The pain may also be produced by setting the patient in a chair and bending him to one side, then forward, and to the other side, which will start the pain as soon as the rib impinges on the edge of the wing of the pelvis. The relief, Clinton stated, is simple and immediate. The end of the offending rib should be dissected for 1 inch, and with it the underlying nerve trunk. Gross specimens show a neuroma-like swelling in the nerve trunk at the point of injury.

Corbett reported three clinical cases in which the condition was diagnosed by him as neuroma in postoperative cases. In the first case the patient suffered almost as much pain after nephrectomy for multiple stones as before. Neurologic examination showed a disturbance of sensation along the course of the ilio-inguinal nerve. A second operation revealed the fact that the nerve had been ligated, with the accompanying artery, and at the point of ligation a lateral neuroma had formed. Cutting the nerve completely relieved the patient's pain. In the second case there was pain in the distribution of the eighth dorsal nerve following resection of the rib and drainage for empyema. At a second operation a neuroma of the eighth nerve was found, which had apparently been caused by the sharp end of the cut rib impinging on the intercostal nerve. The third case was that of a patient with pain in the scar after an operation for benign tumor of the breast.

Corbett stated that in making a diagnosis of neuroma, the following points should be kept in mind: The pain in amputation stumps is referred to some part or parts of the missing limb, and in abdominal cases it is apt to be referred to the distribution of the involved nerve. If section of the nerve is complete, the area of cutaneous distribution will exhibit anesthesia. If the division is partial, hyperesthesia and hyperalgia occur in about the same place. The neuroma itself is tender on pressure. Palpation sends sharp shocks of pain along the course of the nerve, and occasionally the neuroma may be felt. He stated that neurologic examination may reveal lesions of the nerve in patients presenting themselves for reoperation for adhesions.

I am unable to find any satisfactory reference in the foreign literature in relation to this condition.

The following four cases are reported, as I believe that if this condition is brought to the attention of surgeons, many more cases will be detected:

REPORT OF CASES

Case 1.—S. L., aged 30, a nurse, was operated on Oct. 26, 1920, for chronic appendicitis. A right rectus incision was made. On the third day following operation she had so much pain in the abdominal wound that one of the clips was removed. As soon as she was up and about, she began suffering pain to the median side of the scar. This pain was steady, but became much worse when she became tired or nervous. The pain was so severe that in 1923 she reentered the hospital for observation. During this time she had some pain and rigidity in the region of the scar, associated with occasional vomiting. She lost 15 pounds (6.8 Kg.) in weight. While in the hospital her condition was thoroughly studied, and roentgenograms of the gallbladder and gastro-intestinal tract were made, as well as gastric analysis and one duodenal pocket. It was thought that she might have some disturbance of the gallbladder. After leaving the hospital she was able to be up and about, but the pain still persisted—so much so that she took about 30 grains (1.87 Gm.) of acetylsalicylic acid and from 10 to 15 grains (0.65 to 0.97 Gm.) of soluble barbital, U. S. P., or some similar preparation a day, in order to be able to keep on in her work. She had a second severe attack in April, 1925. At that time she had some nausea and vomiting, and tenderness in the region of the scar, with some spasm of the rectus muscle. At this time an exploratory laparotomy was done, through a median incision. There were no adhesions beneath the scar. One small omental band went to the appendix stump. This omental band was freed. The tubes and ovaries and the rest of the viscera were normal on palpation and inspection. When the patient left the hospital, however, the pain still persisted, and she was unable to return to work. In November, 1925, seven months after the exploratory laparotomy, I decided that she must have a neuroma, on account of the marked tenderness localized at the mesial side of the scar. It was decided to inject procaine hydrochloride to determine whether the pain would be relieved thereby. No relief was felt on injection of procaine hydrochloride into the skin or subcutaneous fat, but on injection into the deep fascia, almost immediate relief from pain was noted. This relief persisted for about three days, when the pain gradually recurred. Therefore in November, 1925, the patient was sent to the New York Hospital, and a resection of the abdominal wound, fat and fascia en bloc, down to the peritoneum, was performed. Care was taken not to ligate any nerve that might accompany a blood vessel. A specimen was sent to the laboratory to determine whether neuroma was present, but unfortunately it was left to dry, and hence no satisfactory examination could be made. The convalescence was uneventful. Since that time the patient has had no recurrence of the former pain, and has been able to do severe nervous and physical work without losing a day. She has had none of the former nausea, vomiting or abdominal pain.

Case 2.—R. C., aged 31, was admitted to the Fifth Avenue Hospital on Feb. 20, 1928, complaining of colicky pain in the right upper quadrant. She had had no nausea or vomiting. Eight years previously she had had an appendectomy through a right rectus incision. The pain was in the region of the scar and was exaggerated by the intake of food and by cathartics, and relieved at times by rest. The patient had had some symptoms suggesting hydronephrosis, and pyelograms showed a moderate dilatation of the pelvis of the right kidney. There was no definite evidence of stricture. On examination a well healed

abdominal scar was felt, with no evidence of hernia. There was definite tenderness on pressure to the mesial side of the scar at the upper angle. As the tenderness was so constantly marked in the region of the scar, and as the onset had occurred about one year following the previous operation, it was thought advisable to inject procaine hydrochloride into the fascia at the wound. One-half per cent procaine hydrochloride was used. After injection, the pain was completely relieved for two days. Operation was performed on February 29. An elliptical incision with scar tissue down to the peritoneum was made. A dermal suture was placed over the upper angle of the excised scar for identification in the laboratory. After excision of the scar, the peritoneum was opened and the abdominal cavity inspected. There was some adhesion of the omentum to the old scar. The gallbladder was palpated. It was thin and emptied easily. The kidney was palpated and seemed to be about normal in size, and no thickening could be felt in the region of the pelvis. The kidney could be moved only about 1 inch in a vertical direction. The uterus and ovaries were normal. The abdominal wound was then carefully closed in layers, care being taken to avoid clamping any nerves in the mass.

The pathologic report by E. S. Jessup was as follows: Section of the scar at the point indicated showed dense fibrous tissue and congestion of the vessels. No nerve tissue could be recognized, except a few small bundles near some blood vessels.

On June 10, 1928, the patient reported that the pain in the right lower quadrant had disappeared since operation, and that she could work without any handicap. She had lost the pain over the right hip, had gained 10 pounds (4.5 Kg.) in weight and was not constipated. She had no complaints.

On July 9, 1929, however, the patient complained of pain in the scar and a return of the original symptoms, which had become more marked. She stated that the pain was located in the upper angle of the incision, was tender on pressure and was made worse by coughing. Examination of the scar showed a large keloid, which was tender on pressure at the upper angle.

Comment.—This patient had been treated for a hydronephrosis on the right side due to the pain in the back, while a pyelogram showed only a slight deformity. While the patient was in the hospital, the differential diagnosis between ureteral stricture and neuroma was discussed by the urologic and surgical departments. At the first follow-up, six months after operation, the patient's condition was satisfactory, and she was free from pain. A year later, however, she returned to the follow-up clinic with a keloid formation in the upper angle of the scar and pain in that region. As the eleventh thoracic nerve supplies the region of the incision, reflex nerve stimuli from a neuroma might easily be accountable for the pain she suffered in her back. The return of her symptoms following a keloid formation would seem to indicate that superficial fibrils coming from this nerve had again become caught in this scar.

Case 3.—B. W., admitted to the Lincoln Hospital on April 14, 1927, had been operated on for appendicitis in May, 1925. She complained of pain in the incision not accompanied by nausea; there was no constipation. The pain came on suddenly, especially on exercise, such as walking up stairs.

On physical examination there was tenderness superficially, which did not seem to be interabdominal. The provisional diagnosis was neuroma. On April 18, procaine hydrochloride was injected about the scar. On the following day, the patient stated that the tenderness which she had complained of on admission had almost entirely disappeared. A gastro-intestinal series showed the stomach to be normal in size and shape and to be situated low in the abdominal cavity. The duodenal cap was normal. There were no filling defects or other abnormalities. Examination after six hours showed the stomach to be empty and the head of the barium sulphate column just entering the cecum. An examination after twenty-four hours showed that the transverse colon was situated low in the abdominal cavity, but no other abnormality was noted.

Excision of the abdominal scar was made on April 22, 1927. An elliptical incision was made excising en masse the firm scar down through the interior of the rectus muscle to the peritoneum. The aponeurosis was then united with interrupted chromic and the skin with silk sutures, care being taken not to ligate any nerve accompanying a blood vessel.

The pathologic report was as follows: Section consisted of connective tissue, several thrombotic blood vessels and many fat cells. No nerve tissue was found.

The patient left the hospital free from pain, with a perfectly healed scar. Unfortunately, there was no follow-up in this case.

Case 4.—F. A., aged 16, was admitted to the Fifth Avenue Hospital in May, 1929, complaining of pain in the right lower quadrant. She had been operated on in this hospital sixteen months before for chronic appendicitis, and had made an uneventful recovery. About three months after discharge she began to have pain, mostly in the region of the scar. This was accentuated when she bent over, was more severe at times and was associated with vomiting. The bowels were constipated. The urine was normal. One per cent procaine hydrochloride was injected into the scar and rectus sheath, which relieved the pain entirely for one day.

The scar from the former incision was excised with fusiform incision. This was carried down to the fascia of the external oblique muscle, which was carefully dissected to expose the scar. The small scar in the external oblique was not over the region lateral to the rectus sheath, but was at the margin. A small opening had been made accidentally in the anterior layer of the rectus sheath, and through this the region under the appendix scar was examined. It did not show anything to warrant further exploration in the rectus sheath. The aponeurosis of the external oblique was split in the direction of the fibers inward and outward. The only evidence of the deformed anatomic structures of the former operation was a little irregular fibrous band in the rectus sheath. The outer flap of the external aponeurosis was used to overlap the small defect made in the rectus sheath. The wound was closed with interrupted chromic sutures, the pocket and fat with interrupted plain sutures and the skin with skin clips.

On Oct. 20, 1929, the patient was entirely symptom-free and had been free from pain since the operation in May of that year. The excision of the painful scar had entirely relieved her from all the symptoms. The scar resulting from the second operation was ridged up and slightly wider than one would expect. The patient evidently developed keloid readily; otherwise her general condition was good. She was advised to use mercurial ointment on the scar every other night to determine whether this would have any effect.

COMMENT

After a study of these cases, I am convinced that injuries to the nerve are more common in abdominal wounds than is generally recognized. Moreover one sees numerous cases of diminished tension of muscles when nerves have been cut. While one does not see evidence of definite hernia, one does see relaxed and atrophied muscles which must cause some functional interference. I believe that the methods of operative approach for the abdominal cavity must be reconsidered. The tendency has been to attempt to follow muscle planes, and frequently to sacrifice ruthlessly the accompanying nerves. It is known that muscles heal kindly, even if cross-cut, provided there is no infection. It would therefore seem that surgeons should readjust their ideas to attempt to place incisions so that they run parallel with nerves. If this theory is adopted, how is one to approach the abdominal cavity? The following suggestions are made:

I believe that, whenever possible, incision through the right rectus muscle should be eliminated from the surgeon's armamentorium. After several years spent in a follow-up clinic, I feel firmly confident that this incision is inadvisable for the following reasons: 1. Percentage of hernias after a right rectus incision is higher than after most incisions. 2. Frequently weak wounds are encountered, wherein the portion of the rectus muscle medial to the scar is paralyzed or at least inhibited in action. 3. Injuries to the nerve are more apt to occur when this type of incision is made. In making a right rectus incision one frequently encounters the deep epigastric vessel. There is usually a considerable amount of hemorrhage, and it is impossible to isolate the nerves going to the muscle, so that it is easy to place a ligature about both vessel and nerve. Frequently hematomas result from the injury to the vessel, and in some cases definite thrombosis has been noted along the course of the vessel, extending as far as the femoral vein.

In the upper right rectus incision the transversalis fascia runs directly from the intercostal margin to the mesial line as a well developed layer. It is difficult to close this layer by sutures. When the patient vomits or coughs, the spread of the ribs puts extreme tension on the suture line. Tearing of the fascia is frequently the forerunner of a ventral hernia.

If the surgeon prefers an incision to the right of the median line, either for exploration or for a better approach to the appendix, the Kammerer modification seems preferable to the usual right rectus incision. When the rectus muscle has been drawn to the mesial side, the nerves can be easily identified and retracted up and down, so as to allow a satisfactory exposure. Moreover, if it is necessary to sacrifice a nerve, it can be done under the eye, so that no unnecessary ligature is placed about it. In this type of incision, deep epigastric vessels are not encountered, and therefore there is less hemorrhage (fig. 2).

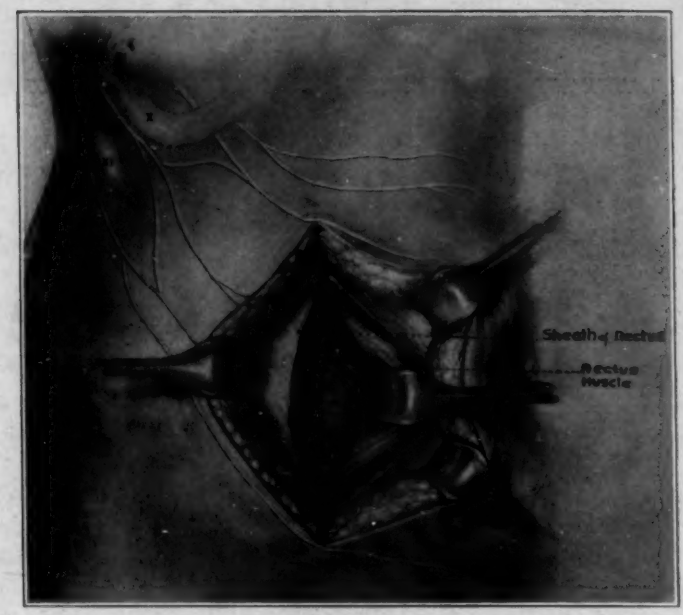

Fig. 2.—The Kammerer incision. The rectus muscle is drawn mesially. The nerves may be seen entering the rectus muscle and can be retracted either upward or downward. If more exposure is needed, they may be cut without ligation.

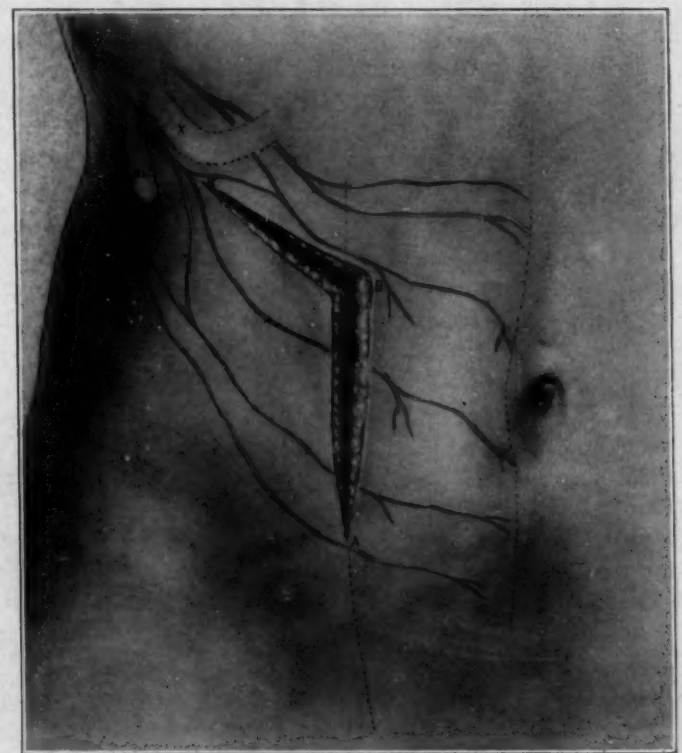

Fig. 3.—Extension of right rectus incision (Kammerer) for exposure of gallbladder. *B'-B* extension is parallel to nerves so that only one nerve is cut during exposure.

Often the surgeon wishes to remove an appendix, and at exploration finds pathologic changes in the region of the gallbladder. The problem then presents itself whether to enlarge the incision—with the possibility of a weak scar—or to close the lower incision and make a second one, with the resultant loss of time. I suggest that if a Kammerer incision has been made, the wound may be extended by a high oblique incision—one parallel to the McBurney incision—which begins at the rectus sheath and ends at the ninth costal cartilage in the mammary line. By this incision it is necessary usually to cut only one nerve, that at the upper angle of the Kammerer incision. This nerve can easily be seen at the first incision, and can be cut without danger of ligating it with the accompanying vessel. The extension of the incision runs parallel to the course of the intercostal nerves. A satisfactory exposure can be obtained thereby (fig. 3).

Upper or lower paramedian incisions are satisfactory, because neither vessels nor nerves of any considerable account are encountered.

The McBurney and the low Pfannensteil incision are ideally planned to avoid trauma to the nerves. The Kocher incision for exposure of the gallbladder involves the risk of injuring nerves, but less so than the right rectus incision.

Transverse abdominal incisions, extending either to or through the rectus muscles, are preferable to lateral vertical incisions, as fewer nerves are encountered and as cut muscles heal satisfactorily after suture.

CONCLUSIONS

1. Injuries to the nerves in abdominal incisions followed by neuromas or neuritis are more prevalent than is generally recognized.

2. They are probably often produced by ligating a blood vessel with its accompanying nerve.

3. In the cases reported in this article the right rectus incision was the procedure followed.

4. Diagnosis of neuroma may be made by testing out the sensory distribution of a nerve and by causing a cessation of the pain by temporarily blocking the nerve by injecting procaine hydrochloride. In right rectus incisions this is accomplished by injecting procaine hydrochloride beneath the fascia of the right rectus muscle.

5. Incisions for abdominal exposure should be planned to avoid trauma to the nerves. Mass ligature of vessels in the vicinity of nerves should be carefully avoided.

THE EFFECT OF ACUTE EXPERIMENTAL CHOLECYSTITIS ON THE EMPTYING OF THE GALLBLADDER *

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Mann,[1] in 1921, first reported the production of specific cholecystitis by the intravenous injection of Dakin's solution (a solution of chlorinated soda). At that time, however, studies were not made on the emptying time of such acutely inflamed gallbladders, as there was no evidence to show that the gallbladder emptied. Many attempts were made to produce chemical cholecystitis by the use of a solution of chlorinated soda subsequent to the discovery that the gallbladder emptied after the ingestion of a meal of fat, but because of variation in the commercial product, positive results were not attained. In the light of present, recently acquired knowledge concerning the emptying of the normal gallbladder following a fat meal, I was prompted to determine whether or not an acutely inflamed gallbladder empties, and to what extent such emptying occurs following the ingestion of a meal rich in fat.

METHOD OF STUDY

In these studies on the production of specific cholecystitis by chemical means, as well as the effect on the emptying of such diseased gallbladders, dogs were used. Pathologic changes were produced within the wall of the gallbladder by the intravenous injection of varying amounts of eusol (a solution of boric acid). Animals were placed on a fast three to four days prior to operation, and explored four hours after the administration of a meal of egg yolk and cream. Through a median line incision, direct observations were made on the degree of cholecystitis and the site of the lesions in the gallbladder, as well as on the emptying of the viscus. The latter data were substantiated by additional observations which will be discussed more in detail under the heading "Operative Procedures." Finally the contents of the gallbladder were aspirated and an equivalent amount of iodized poppy seed oil, 40 per cent, was injected, care being taken not to soil the peritoneal cavity.

SOLUTION AND INJECTION

Animals in good general condition were selected, without any special reference to age, weight, size or sex. Although previous observations on the production of specific cholecystitis by chemical means in which Dakin's solution was used had been made, there still remained certain

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* Work done in Division of Experimental Surgery and Pathology.

1. Mann, F. C.: The Production by Chemical Means of a Specific Cholecystitis, Ann. Surg. **73**:54, 1921.

disadvantages in connection with its use. A solution of eusol originally prepared by the Edinburgh unit during the World War seemed less toxic, the margin of safety seemed greater, thrombosis of the veins was seldom encountered and hemostasis was more easily controlled than when Dakin's solution was used. The solution for injection is prepared by placing 12.5 Gm. of sodium hypochlorite (chlorinated lime, Drackett) and 12.5 Gm. of crystalline boric acid in a large beaker in 1,000 cc. of distilled water. The solution is allowed to remain over night, is filtered once through filter paper the next morning and is ready for use after being warmed slightly to body temperature. All injections were made into the jugular vein. A definite dosage cannot be prescribed for each kilogram of body weight as an assurance of the development of inflammatory lesions within the wall of the gallbladder, and experiment has shown that an animal receiving 30 cc. for each kilogram of body

TABLE 1.—*Varying Amounts of Eusol Injected for Production of Different Degrees of Cholecystitis*

Dog	Weight, Kg.	Eusol, Cc.	Cholecystitis, Grade	Eusol, Cc. for each Kg. of Body Weight
1	23.5	control	control	control
2	22.8	440 (in 2 injections)	1	15.2
3	20.0	400 (in 2 injections)	1	20.0
4	10.4	243 (in 2 injections)	2	23.3
5	12.3	200 (in 2 injections)	2	16.7
6	20.5	500 (in 2 injections)	3	24.4
7	6.0	250 (in 3 injections)	3	41.7
8	10.0	90 (in 1 injection)	4	9.0
9	10.5	135 (in 1 injection)	4	12.8
10	11.8	320 (in 2 injections)	4	27.1
Average	14.8	258 (in 2 injections)	1 to 4	19.0

weight in one or two injections would be certain to reveal definite pathologic changes in the wall of the gallbladder. However, such amounts are seldom necessary, and the risk is too great to chance invaliding the animal and thereby disturbing normal physiologic and metabolic processes. Experimental data in this direction seem to indicate that an average of about 20 cc. for each kilogram of body weight (table 1) would be a good index of the production of such lesions. Furthermore, the rapidity with which the solution is injected into the circulation is significant. Repeated observations have shown that eusol may be injected so slowly in very large quantities that changes in tissue are not produced, or so rapidly that the animal rapidly succumbs during the injection, with or without the production of any lesion of the gallbladder. Although it is not yet certain what the causative agents are in the production of such lesions of the gallbladder after the injection of eusol, it is believed that calcium plays a much more significant part than does chlorine or its associated compounds.

Positive results follow the injection of large amounts of eusol only after considerable experience in this direction. There is without question an individual susceptibility, not only to the toxicity of the solution, but in the production of lesions within the wall of the gallbladder as well. In one animal the lesions may develop after the administration of 40 cc., whereas in another animal of the same weight, sex, age and under the same conditions receiving 400 cc., lesions cannot be produced. Experimental data have shown that the condition of the gallbladder may be prognosticated with a high degree of certainty, not so much with reference to the degree or grade of cholecystitis, as to the presence or absence of lesions in that particular organ. Likewise, as experimental data have accumulated on this subject, it has been shown that a high percentage of inflamed gallbladders can be produced by this method. In the last ten animals injected by such measures, more than 80 per cent revealed lesions of the gallbladder, at least in some degree.

The specificity of Dakin's solution for the gallbladder has been referred to repeatedly in the literature, and undoubtedly the same is true of eusol, as it is a modification of Dakin's solution in which sodium carbonate is replaced by boric acid. Nevertheless, one finds, occasionally, lesions elsewhere in the body, which may be explained by the injection of unnecessarily large amounts of the solution in an attempt to produce a severely inflamed gallbladder. In this series of experiments, however, operation showed that all gross pathologic changes within the abdomen were confined to the gallbladder.

OPERATIVE PROCEDURES

The degree of cholecystitis at the time of exploration was estimated on the basis of grades 1 to 4. Cholecystitis graded 1 consisted of a few small hemorrhagic lesions anywhere in the wall of the gallbladder; cholecystitis graded 2 represented five or six such lesions, and cholecystitis graded 3 or 4 represented widespread hemorrhagic involvement of the entire wall of the gallbladder. It is clear from this classification that although there are only slight variations between grades 1 and 2 or between grades 3 and 4, there are decided differences between grades 1 and 4. Various degrees of cholecystitis are represented in this series, including two each of grades 1, 2 and 3, and three of grade 4, so that certain observations have been made possible. The series of ten animals was divided into three distinct groups, between which only slight variations in the procedure were maintained (table 2).

The three dogs in group 1 were given intravenously 0.05 Gm. of rose bengal for each kilogram of body weight in a 5 per cent solution in distilled water. The animals were then placed on a fast for from three to four days, during which time the dye was permitted to concentrate in the gallbladder. Through a median line incision four hours after the ingestion of a test meal, direct observations were made of the lesions, with notations on the emptying of the gallbladder. The latter data were corroborated by exploration of the duodenum directly above the entrance of the common bile duct and by noting any recent excretion of the dye on the duodenal mucosa. A sudden deposition of the rose bengal on the duodenal mucosa was easily recognized, and confirmed any contraction of the

gallbladder that might have taken place. It is reasonable to assume in this regard that four days were sufficient for the bile ducts to free themselves from any gross trace of the dye. The duodenum was closed in two layers by the Connell suture method. The content of the gallbladder was then aspirated, and an equal amount of iodized oil was injected. The abdomen was closed in four layers in the usual manner, and the animals were returned to the kennels to recover from the effects of the anesthetic. The fast was continued. From six to eight hours later, roentgenograms of the gallbladder were taken, after which a second meal of egg yolk and cream was given. Roentgenograms were then taken at frequent intervals so that any emptying of the gallbladder might be observed.

In group 2 (three dogs), the procedure was the same, except that the dye was omitted and the duodenum was not explored. Such a procedure would tend to eliminate any criticism that the motor activity of the duodenum and its nerve supply had been disturbed.

In group 3 were the remaining four dogs, including one dog which was used as a control. The animals were treated as were those of group 2, except that the mixture of egg yolk and cream was omitted previous to operation, and roentgenograms of the gallbladder were not taken until twenty-four hours after exploration. The slight variations within the last group would remove the possibility of any fatigue of the emptying mechanism of the gallbladder.

TABLE 2.—*Observations in Three Groups of Animals Representative of Various Degrees of Cholecystitis*

		Direct Observations				Roentgen Observations		
Group	Dogs	Fat Meal, 4 hours Previous to Exploration	Rose Bengal	Duodenum Explored	Gallbladder Bile Aspirated; Lipiodol Injected	Cholecystitis, Grade	Fat Meal, Hours After Exploration	Results
1	3	+	+	+	+	1, 2, 4	8	No emptying
2	3	+			+	1, 3, 4	8	No emptying
3	3 and control				+	2, 3, 4	24	Slight emptying in a case of cholecystitis, graded 4

In most instances, the animals prior to operation lapped their meal of egg yolk and cream vigorously and appeared like normal healthy animals. If vomiting occurred that could not be controlled the animal was omitted from the study. Occasionally, a dog had to be encouraged to swallow the egg yolk and cream. This was accomplished either by inserting the tip of a syringe into the buccal fold or by slowly administering the contents by means of a small beaker. The stomach tube was never resorted to for fear of its inhibitory action on the mechanism of the gallbladder.

In every case following the administration of egg yolk and cream previous to operation, the absorption of fat was found to be good and the lymphatic channels, as well as the mesenteric lymph nodes as far down as the ileum, were well engorged. This seems to indicate that the mechanism of absorbing fat had not been disturbed to any decided degree.

Such direct observations did not disclose evidence to indicate that contraction of the inflamed gallbladders might have taken place follow-

ing the administration of the usual fat meal. Exploration of the duodenum in group 1 has substantiated the observations, as rose bengal was never seen on the duodenal mucosa. The observations seem to indicate that contraction of the acutely inflamed gallbladder does not take place following the usual test meal.

An observation made at operation included the rather marked distention of the gallbladder in all animals with cholecystitis graded 1 to 3 inclusive. It is estimated that the gallbladder was increased from a fourth to a third of its normal size. These observations seem to substantiate those of Bollman, Mann and DePage [2] made in 1925, in which they demonstrated that the concentrating function of the gallbladder may be impaired or totally lost in the presence of an acute inflammatory process. However, if cholecystitis was graded 4, the gallbladder was always found to be decreased in size from what would be expected from the size of the animal. Nevertheless, on aspiration the bile did not appear to be thickened or as concentrated as in the normal animal. Also, the biliary tract was found to be dilated in cases of advanced pathologic changes, although changes could not be detected about the cystic or common bile ducts. That the ducts were patent has been confirmed repeatedly both at operation and at necropsy, by pressure on the gallbladder, as well as by histologic examination. Even in the absence of serial sections, it is believed that obstruction to these passages was not present. It may be noted that in not a single instance has jaundice been observed following the production of cholecystitis, even though animals have been observed as long as three weeks after the injection. Therefore, it seems that the cystic and common bile ducts play little, if any, part in this pathologic entity, for at no time did the hemorrhagic exudate extend to that particular region. In some of the more advanced grades of acute cholecystitis, the gallbladders were reduced in size from a sixth to a fourth the capacity of the normal gallbladder. Bile could enter the cystic duct without difficulty, for in some instances of the more advanced grades of chemical cholecystitis, rose bengal was not administered until after the inflammation had been produced, and then there was no apparent difficulty in recovering the dye in high concentration from the lumen of the gallbladder. From examination of gross as well as microscopic sections of such gallbladders, it is difficult to understand how concentration of the bile could occur following such severe inflammation of the wall of the gallbladder. The size and appearance of the gallbladders in cholecystitis graded 4 may be explained, therefore, by the inability of the wall to expand

2. Bollman, J. L.; Mann, F. C., and DePage, P.: The Effect of Specific Cholecystitis on the Bile Concentrating Activity of the Gallbladder, J. Lab. & Clin. Med. **10**:544, 1925.

further because of the severe inflammation rather than by any other peculiarities incidental to the condition.

PATHOLOGIC CHANGES AND COMPLICATIONS

The pathologic changes following the injection of eusol are not much different from those described by Mann following the injection of Dakin's solution. Lesions of the gallbladder appear as small, subperitoneal hemorrhagic areas resulting from marked breaking of the capillaries and infiltration of the wall. They vary in size from 1 to 20 mm. and are situated anywhere over the area of the gallbladder. The wall appears to be thickened, indurated and more or less edematous,

Fig. 1.—Large areas of a hemorrhagic exudate with marked induration, edema and increased thickening of the wall of the gallbladder. A hematoma may be noted at the fundus of the gallbladder.

with a generalized deep purplish discoloration besides the hemorrhagic areas (fig. 1). The superficial vessels coursing about the viscus are always found to be dilated with frequent small areas of a hemorrhagic exudate along their route where the vessel has ruptured. A microscopic examination confirms the gross observations by revealing large areas of hemorrhage. In the less advanced cases of cholecystitis, the hemorrhagic exudate is usually confined to two distinct layers: the subepithelial layer between the mucosa and muscularis, and the perimuscular layer (figs. 2 and 3). In such instances the areas are frequently so large and well circumscribed as to appear like a large blood vessel. The connective tissue and muscle fibers are well spread apart, giving

the appearance of marked infiltrating edema. In the more advanced cases, the exudate does not appear to be limited and is spread throughout the entire thickness of the wall extending from the subepithelial to the subserosal areas. The mucosa has seldom, if ever, been found to be affected (fig. 4). The wall of the gallbladder is always decidedly thickened, the extent of thickening depending on the degree of involvement. In only one instance, in which many observations were made, has hemorrhage ever been encountered within the lumen of the gall-

Fig. 2.—A section through the wall of the gallbladder in mild cholecystitis. The hemorrhagic exudate is confined to two distinct layers; × 150.

bladder. In a few cases, small hematomas about 1.5 cm. in diameter were found within the wall. The liver appeared to be of the fasting type, with a moderate degree of fatty infiltration. On microscopic examination, marked congestion was found. The sinusoidal areas were engorged and distended with erythrocytes, and the cellular infiltration within the columns of cells was only slight.

Occasionally, in certain cases, numerous minute subperitoneal petechial hemorrhages were found at the base of the mesentery involving the entire gastro-intestinal tract. This may occur even in the

absence of a lesion of the gallbladder. Marked reactions of the gallbladder were noted fifteen minutes after the injection of the solution into the blood stream.

The most common complication that may develop after the use of eusol, aside from acute toxemia, is edema of the lungs. The lungs

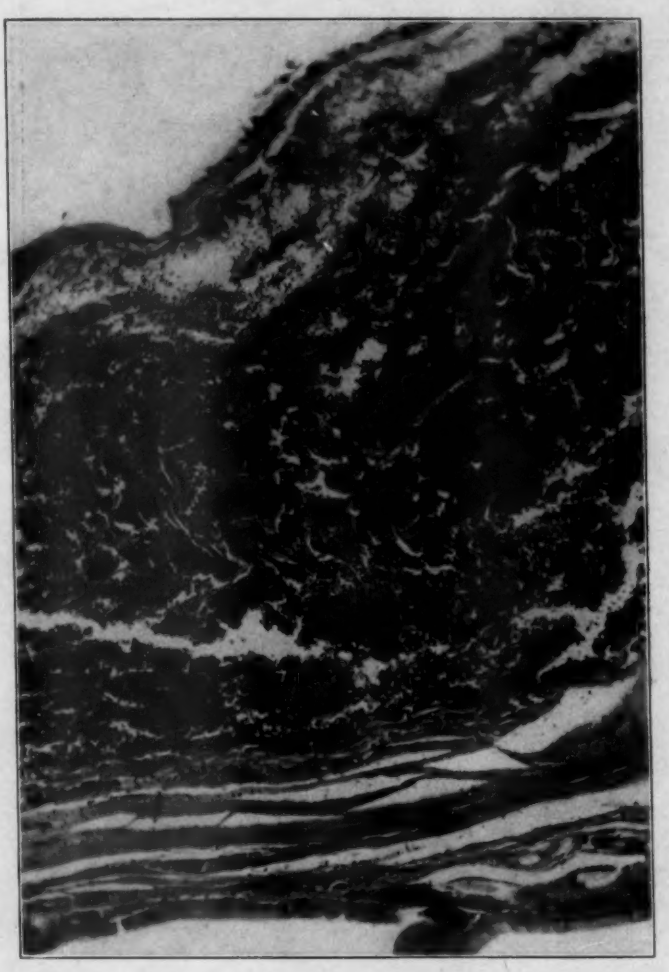

Fig. 3.—A section through the wall of the gallbladder in severe cholecystitis; hemorrhagic exudate extends from the mucosal to the serosal areas; × 150.

become water-logged and large amounts of frothy material are easily expressed. In two instances marked intra-abdominal hemorrhage was found, which resulted from rupture of the large mesenteric vessels. However, hemorrhagic nephritis and hemorrhagic gastro-enteritis so

frequently encountered after the injection of Dakin's solution have never been observed following the injection of eusol. Although there is little question that eusol is the more ideal as compared to Dakin's solu-

Fig. 4.—A section through the wall of the gallbladder in moderate cholecystitis. Absence of any involvement of the mucosa may be noted; × 500.

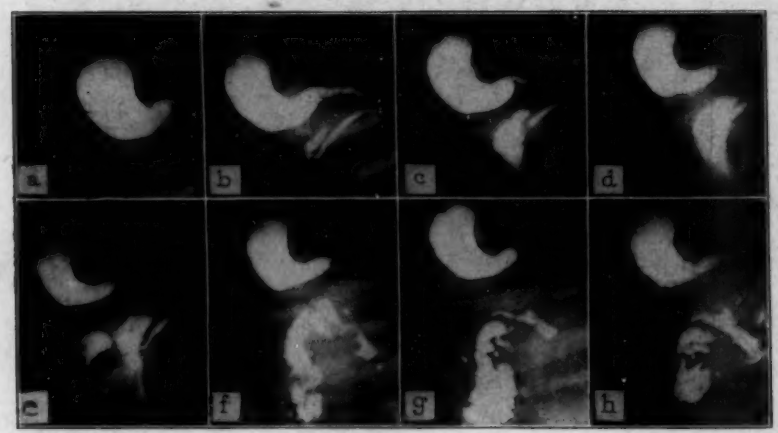

Fig. 5.—The emptying of the normal gallbladder of the dog at frequent intervals following the ingestion of a meal rich in fat; *a*, control; *b*, after five minutes; *c*, after fifteen minutes; *d*, after thirty minutes; *e*, after forty-five minutes; *f*, after one hour; *g*, after two and a half hours, and *h*, after three and a half hours.

tion for the production of such pathologic changes, it has been noted that the gallbladders are practically devoid of change from two to three weeks after the injection (fig. 5).

ROENTGEN OBSERVATIONS

The series of animals was divided into three groups. The first group included the animals which received rose bengal and were subsequently given a mixture of egg yolk and cream four hours prior to operation. The duodenum was explored for any evidence of the dye, the gallbladder was aspirated and an equivalent amount of iodized oil was injected. The animals were then given a second meal of egg yolk and cream from six to eight hours after exploration; animals that

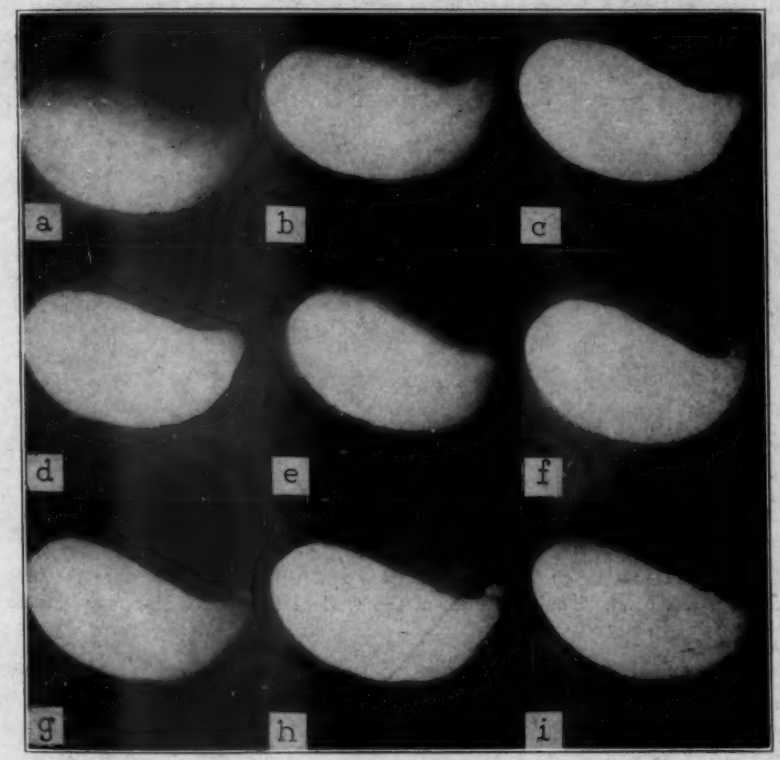

Fig. 6.—A case of cholecystitis graded 4, following the ingestion of a meal rich in fat. Emptying of the gallbladder did not occur; *a*, control; *b*, after five minutes; *c*, after fifteen minutes; *d*, after thirty minutes; *e*, after forty-five minutes; *f*, after one hour; *g*, after two and a half hours; *h*, after three and a half hours, and *i*, after twenty-four hours.

appeared ill or that did not retain the fat meal were discarded. Roentgenograms of the gallbladder were taken at fifteen to thirty minute intervals for from three and a half to four hours, and in some instances even longer. In no instance in the group of gallbladders with lesions graded 1, 2 and 4 was there any evident of emptying (fig. 6). Nevertheless, the

gallbladder showed tonic changes, as evidenced by its contour. Frequently, two hours after the administration of the fat meal, the outline of the gallbladder became decidedly spherical, and its two linear dimensions approached one another. This would seem to indicate that an attempt had been made by the musculature of the gallbladder to reduce its content. These tonic changes, however, disappeared after from ten to twelve hours; the gallbladder then appeared normal but did not show evidence of having emptied. It may be noted that the same cycle is also followed in the gallbladder of the intact animal, and one is able to prognosticate, from early observations of its contour following the ingestion of a fat meal (fig. 7), whether or not such a gallbladder is likely to empty. The original plan was to observe whether the gall-

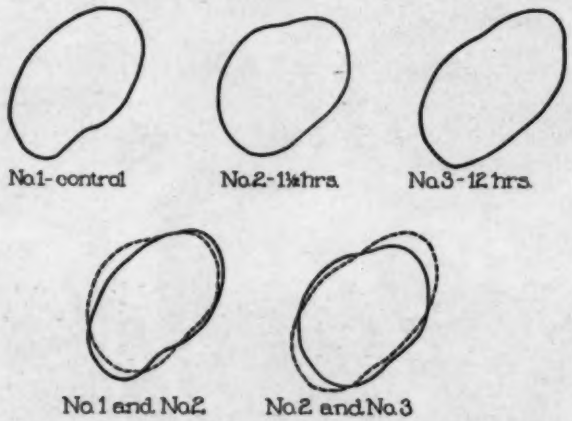

Fig. 7.—A case of cholecystitis graded 2 at frequent intervals after the ingestion of a test meal. Tonic changes of the wall of the gallbladder may be noted.

bladders emptied over a four hour period. Some gallbladders were observed twelve, twenty-four, forty-eight hours and even a week after the repeated administration of a meal rich in fat as well as a regular laboratory diet, and emptying did not occur. This became all the more significant when it was found that after the injection of iodized oil into a normal gallbladder, the gallbladder emptied completely within twelve hours after the animal had been placed on a kennel diet (figs. 8 and 9).

The second group differed only slightly from the first group, and, although egg yolk and cream were administered before and after the operation, rose bengal was not given, nor was the duodenum explored in any case. The group included cholecystitis graded 1, 3 and 4 without other pathologic changes elsewhere in the abdomen. In cholecystitis graded 3 and 4 there was, as usual, no evidence of any emptying. The

characteristic change in the tonicity of the wall of the gallbladder occurred, as had been observed frequently in other animals. In a case of cholecystitis graded 1 in the group in which a few lesions were confined entirely to the fundus of the gallbladder, it was found that one and a half hours after the ingestion of a meal rich in fat, a small amount of iodized oil had been forced out of the cystic duct and into the common bile duct. Three hours later, however, there was only an accumulation within the common bile duct of the iodized oil that had

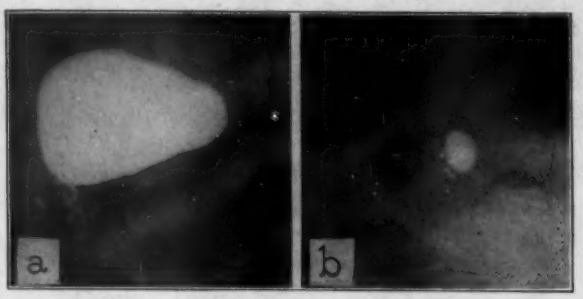

Fig. 8.—Emptying of the normal gallbladder after the dog was placed on the regular kennel diet; *a*, control; *b*, after twelve hours.

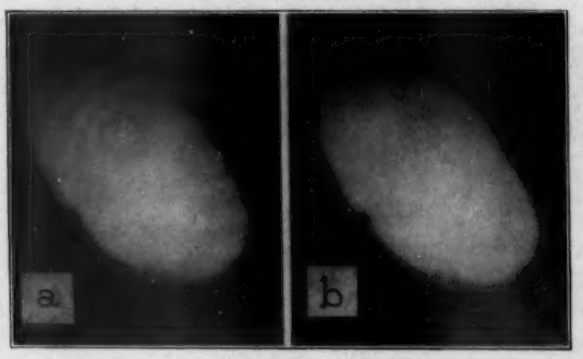

Fig. 9.—A case of cholecystitis graded 3. The animal was placed on the regular kennel diet for one week without showing any evidence of emptying of the gallbladder; *a*, control; *b*, after one week.

previously been expelled. Furthermore, roentgenograms of the gallbladder taken twenty-four hours later, after the animal had been on a kennel diet, showed that, although the gallbladder had completely regained its tonus, there was no indication of additional emptying. It is surprising in this respect how few lesions are necessary to interfere with the normal emptying mechanism in such gallbladders. However, the degree, extent and site of the lesions in this instance may easily

explain the observations. It is not assumed that all normal gallbladders empty completely from three to four hours after the ingestion of a meal rich in egg yolk and cream any more than it is felt that all diseased gallbladders produced by such chemical means do not partially empty under such conditions. It is assumed, however, that most normal gallbladders will show at least some evidence of emptying after a fat meal; on the other hand, only one instance has been encountered wherein a diseased gallbladder as described ever showed evidence of emptying. It must be remembered that occasionally cholecystograms are reported negative as regards lesions of the gallbladder, when at operation the viscus is found to be completely filled with stones. Although the analogy is only slightly divergent, the question of a dependable therapeutic test regarding the presence or absence of a lesion is closely akin, so far as the occurrence of irregularities is concerned.

In group 3, cholecystitis graded 2, 3 and 4 was observed. The animals were explored following the production of the lesions of the gallbladder, the bile was aspirated and an equivalent amount of iodized oil was injected. Twenty-four hours later the animals were given a meal rich in egg yolk and cream, and roentgenograms of the gallbladder were taken at frequent intervals thereafter. Although there was no evidence of any emptying of the gallbladders in which cholecystitis was graded 2 and 3, there were observations in one case of cholecystitis graded 4 in which the gallbladder emptied slightly following the test meal.

SUMMARY

The intravenous injection of varying amounts of eusol has resulted in the production of a high percentage of acutely inflamed gallbladders. The effect of the specific chemical cholecystitis on the emptying of the gallbladder was studied in a series of ten animals. Direct observations, as well as roentgenograms, were made of the diseased gallbladders following the administration of a meal rich in egg yolk and cream. Only one instance has been encountered wherein an acutely inflamed gallbladder, produced by this means, has shown any evidence of emptying. The observations appear to indicate that the acutely inflamed gallbladder does not empty after the usual test meal.

THE MANAGEMENT OF GIANT CELL SARCOMA OF THE VERTEBRAE

REPORT OF A CASE WITH CURE AFTER FIVE YEARS *

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PHILADELPHIA

Bone tumors of the epulis type are generally considered benign. At times, however, the growth invades the contiguous tissues; recurrences are common, metastases being reported by Gross,[1] Coley,[2] Stewart,[3] and others; cachexia is an accompaniment to extension and recurrence (64.7 per cent in the cases reviewed).

While these statements may be considered questionable by those who regard all tumors of the giant cell type as falling in one category, i. e., the benign, they are apparently facts when considered in relation to this type of growth where it invades the vertebrae. The following is an epitome of twenty-eight cases reported in the literature. The average age of the patients was 22.2 years; 46 per cent were below the age of 20 and 80 per cent below 30. Sixty-five per cent of the patients were males. Trauma was mentioned as a causative factor in 33.3 per cent. Pain was severe at the site of the lesion in 55 per cent and was referred in 33.3 per cent. Paralysis of the lower extremities was present in 26 per cent; weakness in 18; anesthesia in 26, and abnormal reflexes in 33.3 per cent. A mass was visible in 75 per cent, and kyphosis was present in 18 per cent. The roentgen diagnosis was correct in 41 per cent of instances, incorrect in 11, and in 48 per cent the roentgenographic report was absent in the protocol.

There were eight deaths, in all, a mortality of 28.5 per cent; two of the patients who died were not operated on; of the twenty-four on whom operation was performed, six died, a mortality of 25 per cent. Three patients died during the immediate operative period; two (cases

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* From the Department of Surgical Research and Pathological Department, Temple University.

1. Gross, quoted by Coley.

2. Coley, W. B.: Ann. Surg. **79**:321 and 561, 1924.

3. Stewart, M. J.: Lancet **2**:1236 (Nov. 28) 1914.

25 and 18) of hemorrhage and one (case 23) of infection; in three of the twenty-four cases in which operation was performed, there were recurrences at the time of death; in the two cases in which operation was not performed, metastasis developed.

RECURRENCES AND METASTASIS

Recurrences.—There was a definite history of recurrence in eleven cases. A follow-up record could not be found in four cases, and four other patients had been under observation too short a time to determine definitely whether or not recurrence would have taken place; the average of the latter was seventy-six days. In those cases in which operation was performed, the minimum time between operation and the patient's discharge from the hospital was twenty-three days and the maximum five months, an average of eighty-five days. As there was a recurrence in case 17 after four years and a definite recurrence in case 2 after four, and probably another after ten years, we believe that the four cases previously mentioned should not be included in estimating the possible percentage of recurrence. If we deduct these eight cases and the three cases that proved fatal during the immediate operative period, there is a percentage recurrence of 64.7 per cent (eleven of seventeen).

Metastasis.—While absolute evidence (microscopic report) that metastasis had taken place was wanting, a review of cases 8 and 9 would seem to indicate that such was the case.

EVALUATION OF THERAPEUTIC PROCEDURES

Exploration of Growth.—In five patients, all of whom had recurrence, the growths were explored; two died as a result of the disease and one was unimproved; mortality, 40 per cent; recurrence, 100 per cent; unimproved, 60 per cent; cured, 40 per cent.

Incomplete Removal.—This procedure was used in eleven cases with the following results: five recurrences and two deaths; no improvement, one case; this shows a mortality of 18 per cent; recurrences, 45 per cent; lack of improvement, 27 per cent; cures, 73 per cent.

Complete Removal.—Complete removal was performed in five cases with the following results: two deaths and three recurrences, with apparent recovery in the latter; this shows a mortality of 40 per cent; recurrences, 60 per cent; cures, 60 per cent.

Complete Removal and Laminectomy.—This procedure was performed in case 14. There was no history of recurrence, but the patient had not improved at the end of eighteen months.

Complete Removal with Subsequent Implantation of Radium at Operative Site.—This procedure was used in case 12. The patient was well at the end of three years, with no recurrence.

No Attempt at Removal; Insertion of Radium Directly Into the Growth.—In a case in which this type of procedure was followed, there were no recurrences. The patient was well at the end of five years. (See case report.)

Erysipelas and Prodigiosus Toxins (Coley's).—This treatment was used in seven cases. In cases 21 and 27 it was the only therapeutic agent used; both patients recovered and had no recurrences. In five, it was used after partial removal of the growth. Three of the five patients had recurrences (cases 4, 5 and 20), but only one was not improved (case 5).

CONCLUSIONS

It would seem that we are justified in drawing the following conclusions from the preceding review:

1. Giant cell tumors of the vertebrae should not be considered benign as compared with giant cell tumors of bone elsewhere.

2. While there is a tendency for them to regress normally they also possess the tendency to metastasize rarely, to recur commonly and to produce cachexia invariably.

3. The proximity of the spinal cord and nerve roots interfere with radical measures (operation). In a percentage of cases the growth begins in the body of the vertebra. This makes complete removal impossible. Regardless of where the lesion originated, removal is usually associated with profuse hemorrhage, necessitating packing, which increases the possibility of infection.

4. Exploratory operation, curettage, partial or complete removal evidently stimulates these abnormal cells to further activity. In over half of the cases (52.4 per cent) there were local recurrences.

5. Part of the high mortality is due to the improper interpretation of the x-ray plate. If the bodies of the vertebrae are affected, extension of the disease is rapid, the intervertebral disks are clear and early symptoms of a lesion of the transverse cord present themselves, one should suspect a malignant condition.

6. The best treatment for this type of tumor is the insertion of radium into or near the growth with as little trauma as possible. Giant cells are peculiarly susceptible to the radium rays; the maximum and minimum doses are appended. The closed method of operation should be used because of the possibility of infection.

REPORT OF A CASE

History.—N. M., a woman, aged 20, had an unimportant family and previous history. She first menstruated when 15 years of age. Her menstrual periods were regular, at twenty-eight day intervals of five days' duration, and painless. There was no change in the character of the flow throughout her illness. At the age of 17, while walking down stairs, she slipped, and in order to regain her balance she quickly threw her shoulders back. This was accompanied by a sharp pain to the left of the lumbar spine, followed by a constant pain in this region, which was so severe at night that it interfered with sleep. It was referred to the posterior and lateral aspects of the left thigh and muscles of the calf of the leg, and confined her to bed for seven days. She stated that a lump appeared to the left of the lumbar spine about the size of a hen's egg about two weeks after the supposed injury. In October, 1924, after three years, she noticed that her left leg was much smaller than the right, and that she was unable to extend her leg and foot; this was associated with a feeling of numbness in the posterior

Fig. 1.—Before operation; the spinous processes of the second and third lumbar vertebrae are displaced to the right; the oval mass is seen below and to the left.

tibial region. During the following month she consulted Dr. L. O. Davis, who advised a roentgen examination.

Physical Examination.—Examination revealed an emaciated woman, aged 20, who weighed 70 pounds. The temperature was 98.6 F., the pulse rate 120. Examination of the head showed no gross abnormalities. The neck was normal. The chest was poorly developed with limited expansion in the apex of the left lung but no abnormal pulsations. The breath sounds were normal, except for questionable râles in the apex of the left lung. The area of cardiac dulness was normal; the heart sounds were strong and regular, and no murmurs were present. The abdomen was scaphoid, with no area of tenderness present, but the recti seemed to have plus tension. No abnormal masses were palpated. Inspection of the postdorsal and lumbar regions revealed a moderately prominent, oval-shaped mass to the left of the lumbar spine, about 8 cm. in the horizontal direction and raised 2.5 cm. from the surface; it was firm and regular in outline, apparently fixed to the spinous processes, and was not tender. The skin overlying the growth was of normal color and moved freely over the mass.

Fig. 2.—Nov. 17, 1924; before operation. The arrows indicate the extent of the tumor, the bodies of the second and third lumbar vertebrae being markedly involved.

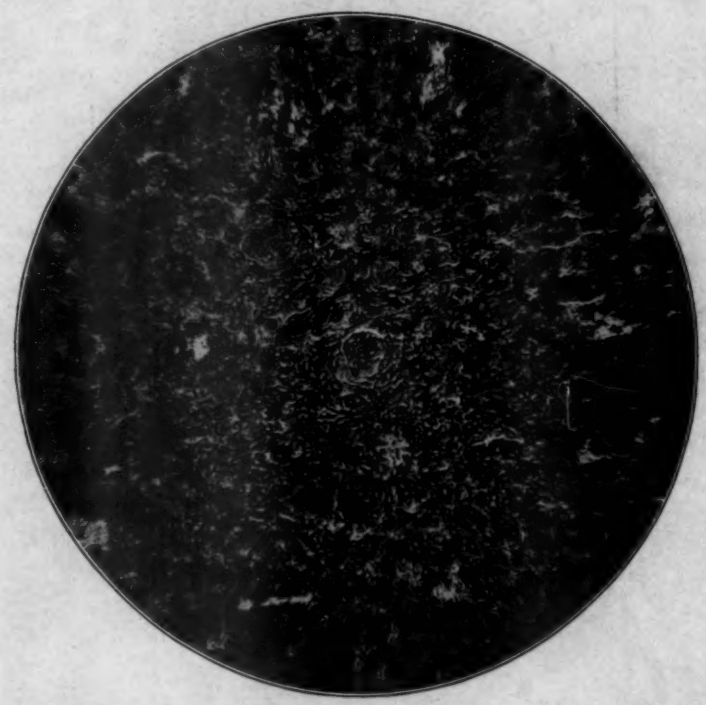

Fig. 3.—High power photomicrograph of the tumor. Note the compact stroma and giant cells with the oval nuclei in the center of the illustration.

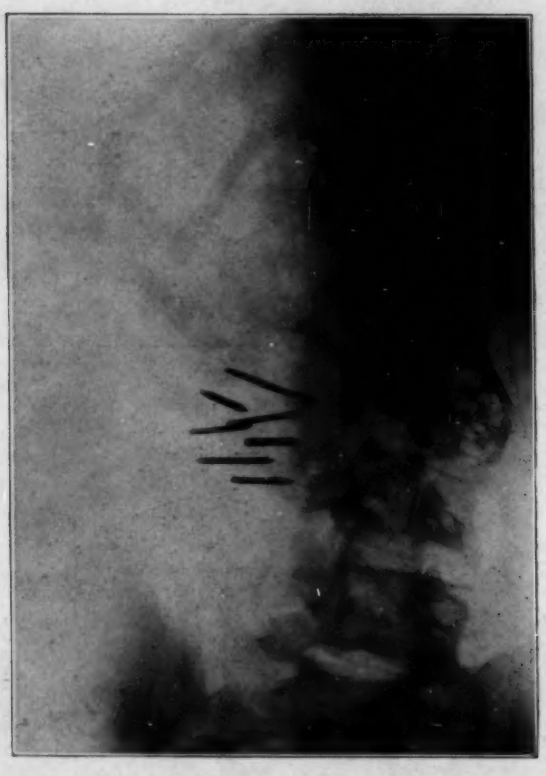

Fig. 4.—Jan. 12, 1925; eight 12.5 mg. radium needles have been placed in the tumor mass about 1.5 cm. distant from the spinal cord. Two insertions were made at six week intervals, making a total of 4,800 mg. hours of radiation.

Fig. 5.—May 22, 1925; four months after the first and three months after second radium implantation. Note the increased density and diminution in the size of the mass.

Fig. 6.—June 21, 1928; three years and five months after the first implantation.

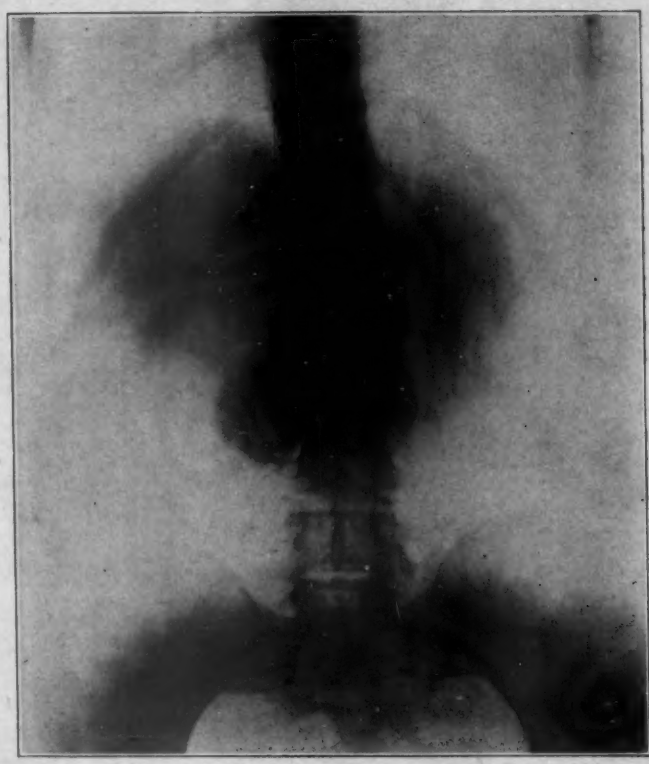

Fig. 7.—March 8, 1929; four years and three months after implantation. Further diminution in the size of the growth with increased density is seen. The patient is married and attending to housework.

Case	Reporter and Reference	Age and Sex	Previous History: Trauma or Tuberculosis	Duration of Pain	Referred Pain	Paralysis	Anesthesia	Mass
1	Manheimer, E.: Beitr. z. klin. Chir. **72**: 741, 1911	16 F	Not mentioned	Not mentioned	Lower extremities	No cord involvement	Not mentioned	Size of a fist at level of second lumbar vertebra
2	Sick: Personal communication to Manheimer	M	Not mentioned	Not mentioned	Sciatic nerve and left lumbar region	Not mentioned	Not mentioned	Fluctuating tumor between iliac crest and twelfth dorsal vertebra on abdominal side; mass had a bony capsule
3	Ashurst: reported by Dean Lewis	14 M	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Right loin, 6 weeks' duration
4	Harmer: Boston M. & S. J., 1915, p. 61	16 M	History of trauma; 3 months before entrance to hospital patient fell striking spine	Pain and tenderness	Lower dorsal region and to occiput	Weakness in right knee	Loss of sensation over exterior and anterior surface of right thigh, 2 weeks	Size of hand in the subcostal region of the tenth dorsal vertebra
5	Mixter: reported by Dean Lewis	31 M	No history of trauma	Girdle	Not mentioned	Weakness in legs; paralysis with sphincter disturbance below fifth dorsal vertebra	Anesthesia in legs, later below fifth dorsal vertebra	Not mentioned
6	Follis reported by Dean Lewis	40 M	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Fluctuating mass between the sixth and twelfth dorsal vertebrae
7	Kesmodel: Am. J. Roentgenol. **8**: 573, 1921	43 M	No	12 months; back not relieved by rest	Pain in left leg	Weakness, atrophy of left leg	Not mentioned	Not mentioned
8	Basch: Hygiea **84**: 769 (Oct.) 1922	31 F	Not mentioned	No early pain	Limbs felt heavy and clumsy	Spontaneous twitching of legs	8 months after onset in abdomen and hips, gradually progressing downward	Fluctuating mass at tenth dorsal vertebra

Kyphosis	Reflex	X-Ray	Operation	Post-operative Irradiation	Pathologic Report	Outcome and Comment
Not mentioned	Negative	Destruction of transverse process of second lumbar vertebra	Tumor removed as thoroughly as possible	Not mentioned	Osteoid spindle cell sarcoma with myxomatous areas containing giant cells	Observed only 5 months; treated with 0.01 Gm. of atoxyl intravenously every other day for 46 days; appeared well at the end of 5 months
Not mentioned	Not mentioned	Distinct shadow between iliac crest and twelfth dorsal vertebra	Partial removal with no improvement; potassium arsenate given; patient well 4 years later	Not mentioned	Giant and mixed cell fibrosarcoma	Recurrence after 4 years; 10 years later, patient alive but not well
Not mentioned	Not mentioned	Involvement of fourth and fifth lumbar vertebrae; roentgenogram 17 months after cessation of treatment revealed shadow of dense bony tissue	Incision; wound packed with gauze; mass increased in size after operation; broke down 9 months later, then because of bony hardness	Not mentioned	Giant cell sarcoma	Coley's fluid given on second, third and fourth days; increased to 2.6 cc. at a dose; accepted for army later
Depression of tenth dorsal vertebra	Ankle clonus; Oppenheim's reflex on right side	Showed recurrence after second operation; negative before	June 1912, exploratory incision revealed a bluish rather tense but fluctuant mass beneath muscles of the back; removed and wound packed with gauze	Not mentioned	Giant cell sarcoma	Treated with mixed toxins for 7 months; injections every other day, then twice each week; reaction severe; rapid recurrence of the tumor 5½ by 3½ by 2 inches; associated paresis of right leg; June, 1913, no mass; June, 1914, another operation because of recurrence; bone cyst found; patient well 2 years later
Not mentioned	Not mentioned	Negative	First, incomplete removal; Coley's toxins given; complete paralysis; recurrence with complete block of spinal canal; laminectomy of fourth and fifth dorsal vertebrae	Yes	Giant cell sarcoma	Recurrence; final outcome not mentioned
Not mentioned	Not mentioned	Not mentioned	Complete removal with recurrence in 4 months larger than first	Not mentioned	Giant cell of epulis type	No follow-up
Lumber scoliosis	Knee jerks absent on left	Negative except for slight hypertrophic changes	Complete removal	Not mentioned	Fibrosarcoma with giant cells	Follow-up not mentioned
Kyphosis of eighth and ninth dorsal vertebrae; tenth depressed	Patella on right absent	Diagnosis of tumor of tenth dorsal vertebra; no treatment	First, exploratory incision showed necrosis	Not mentioned	Giant cell tumor with transverse myelitis	Death; lumbar puncture bloody; air insufflation of spine; questionable metastases in pleural cavity

Case	Reporter and Reference	Age and Sex	Previous History: Trauma or Tuberculosis	Duration of Pain	Referred Pain	Paralysis	Anesthesia	Mass
9	Kono, N.: Sei-i-Kwai M. J. Tokyo, **44**: 18, 1925	29 M	Not mentioned	Rapid development, 6 months	Not mentioned	Not mentioned	Not mentioned	Mass the size of a child's head
10	Madelung: München. med. Wchnschr. **56**: 479, 1909	19 M	Trauma; struck in back 14 years previously	Not mentioned	Not mentioned	Yes	Yes	Not mentioned
11	Shumann: reported by Dean Lewis	27 M	Not mentioned	Pain in back, 3 months	Not mentioned	Paraplegia	Not mentioned	Second lumbar vertebra
12	Lewis, Dean: J. A. M. A. **83**: 1224 (Oct. 18) 1924	7 F	Not mentioned	Night cries for 3 weeks	Stomach	Wabbling gait; weakness of muscles; lower extremities	No anesthesia	Not mentioned
13	Kesmodel, Karl F.: Am. J. Roentgenol. **8**: 573, 1921	16 M	Fell 3 months previously	3 months	Backache	Yes	Over right thigh	Swelling; depression over the tenth dorsal to the right of vertebra
14	Cushing: reported by Dean Lewis	13 F	Not mentioned	Not mentioned	Not mentioned	Both legs; iliopsoas	Below eleventh dorsal vertebra; epicritic 2 inches above	Between tenth and eleventh dorsal vertebrae
15	Cushing: reported by Dean Lewis	16 M	Both	Lower dorsal and lumbar vertebrae	No referred pain	Redness of legs	Not mentioned	Tender 12 months after injury
16	Cushing: reported by Dean Lewis	13 F	Not mentioned	6 months, lumbar vertebra	Left leg and foot, then both legs	Not mentioned	No anesthesia	Not mentioned
17	Madelung: reported by Dean Lewis	25 M	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Mass from twelfth dorsal vertebra to iliac crest
18	Thompson: reported by Dean Lewis	24 M	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Fungating mass over sacrum

Cell Sarcoma of the Vertebrae—Continued

Kyphosis	Reflex	X-Ray	Operation	Post-operative Irradiation	Pathologic Report	Outcome and Comment
Not mentioned	Not mentioned	Not mentioned	No	Not mentioned	Rich in giant cells; also spindle cells, part polymorphonuclears	Death; metastasis in ribs bilateral; chondromyxoendothelioma, probably metastatic development from parotid in temporal region; 10 years' duration; size of hen's egg; reduced by radium; not a metastasis from vertebrae
Not mentioned	Not mentioned	Not mentioned	Curettage of third and fourth cervical cavity size of hen's egg	Not mentioned	Giant cell myeloid sarcoma	Well 3 months after curettage; complete disappearance of symptoms
Not mentioned	Not mentioned	Not mentioned	Complete removal with recurrence of tumor in 8 months, size of man's head; patient readmitted 9½ years later ossification had taken place	Not mentioned	Giant cell	Bladder and rectal disturbance persisted; parapiegia
Not mentioned	Bilateral Babinski sign; ankle clonus; no abdominal reflex	Rarefaction of right half of body of fourth dorsal verteba	Laminectomy of second, third, fourth and fifth dorsal vertebrae; mass removed; dura not opened; 4 weeks later 50 mg. of radium placed for 18 hours in region formerly occupied by tumor; 900 mg.	X-ray same time	Friable vascular mass extending 2 inches along cord; giant cell sarcoma	After 3 years child apparently normal
Not mentioned	Ankle clonus; Oppenheim's reflex	No preoperative roentgen examination	Exploratory opreation; encapsulated liquid containing broken down tissue	Not mentioned	Giant cell sarcoma	No improvement following operation
Not mentioned	Lost knee jerks; achilles jerks present on both sides, but plus on the left	Not mentioned	Laminectomy and extirpation	Not mentioned	Giant cell sarcoma-extradural spindle cells	Automatic bladder and rectum protective reflex; 9 months after operation no voluntary return of paralyzed muscles but reflex present; incontinence; 18 months later sensation and motion had not returned
Not mentioned	Not mentioned	Roentgenogram positive; third to fifth lumbar vertebrae	Partial removal of tumor at third to fifth lumbar vertebra; tumor passed off to right	Yes	Giant cell bony tissue between cartilage and bone	No follow-up
Not mentioned	Not mentioned	Not mentioned	Partial removal of tumor in sacral region	Not mentioned	Giant cell	Death 4 years 4 months after operation; rectal disturbance, 6 weeks; loss of weight and urgency
Not mentioned	Not mentioned	Not mentioned	Extirpation	Not mentioned	Giant cell myxomatous tissue	Recurrence from 4 to 6 years after operation; no metastases but local recurrences; death
Not mentioned	Not mentioned	Not mentioned	Tumor removed; hemorrhage profuse	Not mentioned	Giant cell	Death

Case	Reporter and Reference	Age and Sex	Previous History: Trauma or Tuberculosis	Duration of Pain	Referred Pain	Paralysis	Anesthesia	Mass
19	Leahe: reported by Dean Lewis	Not mentioned	Yes, trauma 5 years previously	Not mentioned	Not mentioned	Left leg	Not mentioned	No mass
20	Greenough, Simmons and Harmer	16 M	Trauma 3 months before symptoms	3 months	Not mentioned	Not mentioned	Not mentioned	Thickening
21	Harmer, T. W.: Brit. M. J. **172**: 443, 1915	21 M	Not mentioned	Not mentioned	Not mentioned	Legs, rectum and bladder	Not mentioned	Yes
22	Reported by Thomas: Brit. M. J. **172**: 443, 1915	F	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned
23	Klebs: Die allgemeine Pathologie, 1889 part 2, p. 732	23 F	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned
24	Volkmann, R.: Deutsche med. Wchnschr.,1876, p. 281	23 F	Not mentioned	Sacrum and rectum 1 year	Not mentioned	Weakness	Not mentioned	Mass of 9 months' duration; for past 6 months, size of egg
25	Gussenbauer, C.: Teischr. f. Heilk. **11**: 473, 1890	33 F	Not mentioned	Continuous sacral pain after childbirth	Not mentioned	No	Not mentioned	Sacral vertebrae
26	Peham, H.: Deutsche Wchnschr. **45**: 241, 1897	17 M	Not mentioned	Sudden pain in sacral region	Not mentioned	Not mentioned	Not mentioned	Mass in sacral region
27	Colby: reported by Dean Lewis	21 M	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Not mentioned	Sofa pillow
28	Bower, Clark and Davis	20 F	Yes, 3 years previously	3 years	To posterior tibial region	Incomplete paralysis of muscles supported by sciatic nerve	No	Mas to left of lumbar spine

Cell Sarcoma of the Vertebrae—Continued

Kyphosis	Reflex	X-Ray	Operation	Post-operative Irradiation	Pathologic Report	Outcome and Comment
Not mentioned	Not mentioned	Not mentioned	No operation	Not mentioned	Necropsy showed a hemorrhagic mass between first and second lumbar vertebrae, which developed from transverse processes	Seven months prior to death patient lost control of bladder and rectum; bed sores developed; death
Not mentioned	Not mentioned	Small tumor of third lumbar vertebra arising from lateral surface of spinous process	Partial excision	Not mentioned	Giant cell tumor; many small giant cells; stroma, fibrous type	Local recurrence in wound immediately; received Coley's toxin treatment by injection direct into tumor for 7 months, 62 injections; tumor sloughed out, wound healed in 2 years; later tumor appeared in scar; no tumor tissue found; well 8 years after operation
Not mentioned	Not mentioned	Not mentioned	No operation; mass extended from eighth dorsal to third lumbar vertebrae, 0.3 inches either side of vertebrae	Not mentioned	Giant cell sarcoma	Treated as Pott's disease for several months; Coley's toxin given 3 months with intervals of rest; maximum dose, 3 minims; patient entirely well from 7 to 8 months later; no recurrence after 12 years
Not mentioned	Not mentioned	Not mentioned	Partial extirpation	Not mentioned	Giant cell sarcoma; tumor of sacrum	Well 1½ years after; Coley's fluid given
Not mentioned	Not mentioned	Second cervical vertebra; began left lateral process	Exploratory	Not mentioned	Giant cell sarcoma	Death in few days, from sinus thrombosis and purulent meningitis
Not mentioned	Not mentioned	Not mentioned	Removal complete	Not mentioned	Giant cell sarcoma	Patient discharged 6 weeks later complaining of painful defecation which gradually subsided
Not mentioned	Not mentioned	Not mentioned	Attempted removal	No	Giant cell sarcoma	Died from hemorrhage during operation; tumor involved fifth lumbar vertebra; no metastases
Not mentioned	Not mentioned	Not mentioned	Exploratory operation for suspected caries third sacral vertebra	Not mentioned	Giant cell sarcoma	Discharged 23 days after operation; no follow-up
Not mentioned	Not mentioned	Not mentioned	No operation	Not mentioned	Giant cell sarcoma	Only treatment, Coley's fluid given; well 2 years later
Yes	Patellar reflex absent; Babinski ?	Sarcoma of bodies of second and third lumbar vertebrae	Biopsy and insertion of radium needles	Surface application of radium	Giant cell sarcoma	In good health after 5 years

The right lower extremity was normal. The reflexes were present, and there were no areas of anesthesia. The muscles of the left lower extremity were markedly atrophied, particularly the posterior tibial group. A foot drop was present. The patellar reflex was absent, and there was a questionable Babinski sign. There were no areas of hyperesthesia or anesthesia. Heat and cold were recognized on any part of the extremity. There was paralysis of the quadriceps extensor, tibialis anticus, extensor longus digitorum and extensor longus hallucis and marked weakness of the hamstrings and posterior tibial group.

Urinalysis gave negative results. Examination of the blood revealed: hemoglobin, 62 per cent; erythrocytes, 3,790,000; leukocytes, 7,100; polymorphonuclears, 74; small lymphocytes, 24; large lymphocytes, 1; transitionals, 1.

The roentgen examination by Dr. G. C. Bird showed an evidently sarcomatous mass involving the bodies of the second and third lumbar vertebrae.

On Nov. 28, 1924, with the patient under gas anesthesia, a 10 cm. semilunar incision was made, beginning at the upper border of the spinous process of the first lumbar vertebra and extending downward to the left and to the upper border of the fourth lumbar vertebra. The skin flap was reflected, the lumbar fascia divided and the erector spinae muscle retracted. The transverse processes were softened and replaced by a bluish-red calcareous material which bled profusely. The mass was about 7 by 5 by 3 cm., the center of greatest density being opposite the spines of the second and third lumbar vertebrae; the pathologic process had practically accomplished a transversectomy. A biopsy was performed, and the pathologic report was as follows: Several small pieces of soft reddish tissue and a few spicules of bone were seen. The section showed a stroma of spindle cells of varying density inclosing numerous thin-walled capillaries and blood spaces unlined by endothelium. Many giant cells were present in the stroma, characterized by an acidophilic cytoplasm and averaging from ten to twelve clear oval nuclei centrally located. A diagnosis of giant cell sarcoma was made.

Eight needles, each containing 12.5 mg. of radium, were inserted into the center of the mass. The fascia was closed with interrupted chromic catgut, the skin with interrupted horse hair sutures. A roentgenogram was made with radium needles in situ to determine the exact position of the element in relation to the cord. The roentgenogram showed them to be from 0.5 to 1 cm. distant. The needles were removed in twenty-four hours, a total of 2,400 mg. hours of radiation having been given. The wound healed primarily; the sutures were removed on the fifth day. Two days after operation, contraction of the tibialis anterior was noted. A plaster of paris cast was applied on the seventeenth day after operation, and the patient was discharged from the hospital on the twenty-first day. Improvement was rapid, and on her return to the hospital on February 8, there was complete return of power in all the paralyzed muscles, and the reflexes were normal. She had gained about 4 pounds (1.8 Kg.). The pulse rate was 112, the temperature normal. The urinalysis gave negative results. The blood count was: hemoglobin, 62 per cent; erythrocytes, 3,460,000; lymphocytes, 8,400; polymorphonuclears, 72; small lymphocytes, 26; large lymphocytes, 1. On February 9, the same operative approach being used, eight 12.5 mg. needles were again inserted into the growth for twenty-four hours. A roentgenogram was made with the needles in position. The mass was smaller, partially calcified and not as hemorrhagic as at the previous operation. The wound healed normally, and the patient was again discharged from the hospital twelve days after the operation. A cast was not applied. A roentgenogram of the chest was negative. The patient continued to improve, and was again admitted to the

hospital on May 21, 1925, at which time radium was applied to the mass 2 cm. distant from the growth, 100 mg. for twenty hours, making a total of 2,000 mg. This was repeated on Aug. 25, 1925. A roentgenogram at this time showed that a large mass of osseous tissue had replaced the tumor mass. The last application of radium was made on Feb. 11, 1926 (2,000 mg.). The patient's general condition was excellent. The contraction of the previously paralyzed muscles was practically normal, and there was but a slight degree of atrophy present. She had no pain. The pulse rate was 100.

Subsequent History.—Five years and one month after the onset of symptoms and five years after radium implantation, the patient is in good health. Disregarding the advice of her family physician, she was married (September, 1929) and is doing her own housework.

NOTE.—Since this manuscript went to press, our attention has been drawn to an article by A. W. Adson (Osteitis Fibroas Cystica of the Spine, Surg. Gynec. Obst. **46**:684, 1928) dealing with two cases in which pulpy, friable spinal tumors developed apparently following trauma. These cases were reported three years and three months, respectively, after the tumors were removed successfully.

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THE EFFECT OF SPINAL ANESTHESIA ON THE CARDIAC OUTPUT *

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The increasing recognition of spinal anesthesia as the method of choice in many operations has seemed to warrant an investigation into its physiologic effect. In this paper we present the results of a series of studies along this line. Since clinical experience has demonstrated that under spinal, as under other, anesthesia the state of the circulation is of primary concern to the surgeon, the first observations have dealt with the cardiac output and blood pressure.

Dogs were used in the experiment. One to two hours before the experiment was begun, morphine (from 10 to 15 mg. per kilogram of weight injected subcutaneously) or barbital (0.3 Gm. per kilogram, intravenously) was given. Arterial pressure was measured by a mercury manometer, the cannula being inserted in the carotid artery. The cardiac output was determined according to the Fick principle, the technic being that employed by Harrison and Leonard.[1] The rubber mask designed by Blalock [2] was used to connect the animal to a Benedict spirometer. The Roth [3] recording device was used. Arterial blood was obtained from an exposed femoral artery; venous blood, from puncture of the right ventricle, as first done by Barcroft, Boycott, Dunn and Peters.[4] The oxygen contents of arterial and venous blood were measured in the Van Slyke-Neill [5] apparatus.

After the control observations had been made, the animal was turned on its side, and spinal puncture was done. The needles and technic were essentially the same as those used for this procedure in man. At first some difficulty was encountered in entering the spinal canal, but after a little practice the puncture was easily performed. It

* Submitted for publication, Feb. 3, 1930.

* From the Departments of Gynecology and Medicine, Vanderbilt University.

1. Harrison, T. R., and Leonard, B. W.: J. Clin. Investigation **3**:1 (Oct.) 1926.

2. Blalock, A.: J. Lab. & Clin. Med. **12**:378, 1927.

3. Roth, P.: Boston M. & S. J. **186**:457, 1923.

4. Barcroft, J.; Boycott, A. E.; Dunn, J. S., and Peters, R. A.: Quart. J. Med. **13**:35, 1919.

5. Van Slyke, D. D., and Neill, J. M.: J. Biol. Chem. **61**:523, 1924.

was usually necessary to employ suction with a syringe in order to be certain that one had entered the cerebrospinal space, as spinal fluid frequently failed to drip from the needle. A solution of 1 per cent procaine hydrochloride and 0.1 per cent strychnine sulphate was used in some experiments and procaine in others. The doses used in most of the experiments were of the same order of magnitude as those employed clinically. In the later observations, the effect of larger doses was studied. The foot of the table was elevated in some experiments, but not in all of them. After the characteristic fall in blood pressure had developed, the cardiac output was again measured.

The results of these studies are shown in the table. No constant changes were observed in oxygen consumption or in the oxygen con-

Effect of Spinal Anesthesia on Dogs

Experiment Number	Control Period: Before Spinal Anesthesia		After Spinal Anesthesia		Percentage Change	
	Minute Cardiac Output	Mean Arterial Pressure	Minute Cardiac Output	Mean Arterial Pressure	Minute Cardiac Output	Mean Arterial Pressure
3	2180	84	(a) 1980	60	— 9	—29
			(b) 1580	28	—27	—67
5	2720	138	(a) 3060	96	+12	—30
			(b) 2540	110	— 6	—20
7	2820	108	2220	66	—21	—39
8	2830	120	2100	70	—10	—42
10	1680	100	1340	66	—15	—34
11	1500	104	660	46	—56	—56
12	1140	116	800	84	—30	—28
13	1300	114	420	34	—68	—90

tents of the blood, and therefore the values for these functions are not presented. As can be seen in experiments 3 and 5, the blood pressure fell before the cardiac output decreased. In six of the eight experiments, the degree of diminution in blood pressure was greater than that in the cardiac output. When the decline in blood pressure was relatively slight (experiments 3a, 5a and 5b), the minute cardiac output usually remained at or nearly at a normal level. (Changes of 10 per cent or less cannot be regarded as significant, as this is approximately the limit of error of the method). Greater diminution in arterial pressure (experiments 7, 8, 10 and 12) were usually followed by moderate decrease in the blood flow, and when the drop in arterial pressure was extreme (experiments 11 and 13) the cardiac output was also very low.

From the data it appears that in spinal anesthesia the initial change is, as would be expected, in arterial pressure, and that the venous return and output of the heart are affected secondarily. This sequence of events is just the opposite of that in hemorrhage. In this condition,

Blalock [6] has shown that the cardiac output decreases first and is already reduced by from one-third to one-half before a significant decline in mean arterial pressure occurs. These observations probably explain the well recognized clinical fact that a low blood pressure due to hemorrhage is a much more serious sign than a similar blood pressure in a patient under spinal anesthesia. These observations lend support to the views of Blalock,[7] who studied the effect of trauma on the spinal cord and found that a given lowering of blood pressure was accompanied by less diminution of cardiac output than was the case after hemorrhage. The same author [8] has recently shown that secondary shock due to muscle injury can be explained almost entirely by local hemorrhage within the traumatized area. It appears that the lowering of the blood pressure under spinal anesthesia is analogous to that occurring in primary shock or collapse or in ordinary syncope. Such an understanding of the mechanism of spinal shock may have therapeutic significance. *A priori,* since the peripheral resistance is primarily at fault, one would expect more value from vasoconstrictor drugs than in hemorrhage, a condition in which the circulatory blood volume, venous return and cardiac output are essentially affected.

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6. Blalock, A.: Mechanism and Treatment of Experimental Shock, Arch. Surg. **15**:762 (Nov.) 1927.

7. Blalock, A., and Bradburn, H. B.: Trauma to Central Nervous System. Its Effects on Cardiac Output and Blood Pressure, Arch. Surg. **19**:725 (Oct.) 1929.

8. Blalock, A.: Experimental Shock: The Cause of Low Blood Pressure Produced by Muscle Injury, to be published.

THE ETIOLOGY OF INTERSTITIAL AND MEDIASTINAL EMPHYSEMA

(EXPERIMENTAL PRODUCTION OF AIR EMBOLISM, ACUTE PNEUMOTHORAX, ACUTE PNEUMOPERITONEUM, INTERSTITIAL, MEDIASTINAL AND RETROPERITONEAL EMPHYSEMA) *

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The term mediastinal and interstitial emphysema in our work is used to include not only the introduction of air into all the areolar tissues of the body, but also acute pneumothorax, acute pneumoperitoneum and air embolism. This condition has often been referred to as surgical emphysema because a good proportion of clinical cases develop after an injury which is usually treated by the surgeon.

The causes of interstitial emphysema as given by various authors may be summarized under the following groups:

1. Traumatic, namely, due to an injury of the lung produced by stab wound of the wall of the chest, a fractured rib or a needle during the induction of an artificial pneumothorax.

2. Sudden increase of the intrapulmonic air pressure resulting from physical strain such as coughing, childbirth or obstruction of the air passages by aspiration of foreign bodies.

3. Entrance of air into the fascial planes during operative procedures that cause an opening of the fascial planes and under certain conditions that allow the air to be sucked in.

4. Retroperitoneal emphysema caused by a perforation or an injury to the retroperitoneal surface of the colon or the duodenum or extension through the diaphragmatic openings from the mediastinum to the retroperitoneal areolar tissue.

5. Administration of general anesthesia by the closed method or the inflation method, in which the intrabronchial air pressure is increased. Lilienthal [1] noted such an emphysema in a patient who received a general anesthetic by the insufflation method, and in whom the air pressure was raised to 60 mm. of mercury.

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* Submitted for publication, Dec. 31, 1929.

* From the Department of Surgery, College of Medicine, University of Illinois.

1. Lilienthal, H.: Thoracic Surgery, Philadelphia, W. B. Saunders Company, 1926, vol. 1, p. 250.

Our interest in this work was aroused by an accidental production of the condition in a dog in a series of experiments on the etiology of pulmonary abscess.[2] As we were introducing the infected blood and sputum into the lung through the bronchoscope, we noted that by the introduction of air into the lungs under suddenly increased pressure an interstitial emphysema was produced. This condition was repeated at will in dead as well as in live animals, and our results were uniformly positive. A careful study of the various underlying factors was therefore undertaken.

METHOD

A small tube was inserted into the mouth or the trachea of a dog under ether anesthesia, and the air was blown in at known pressures. The tracheal tube was large enough in diameter to allow the excess air to return to the mouth and come out. In order to produce a sudden increase of intrapulmonic pressure, we compressed the floor of the mouth or the trachea, and for a known time we held the air in the lung. This sudden increase of pressure, though momentary, was sufficient to cause interstitial emphysema. In a series of experiments, the air was introduced through one source and removed at a different outlet. Under such conditions the outgoing, as well as the ingoing, air pressure could be under control and thus various factors could be studied. By means of a capillary microscope, Dr. D. M. Olkon studied the capillary circulation of the skin under varying degrees of increased intrapulmonic pressure.

EXPERIMENTS AND RESULTS

In the first series of experiments we made an effort to find out what happens when a blast of compressed air is blown into the lung. With the dog under ether anesthesia, air was allowed to enter the trachea through a bronchoscope that was passed beyond the larynx. With a compression of the trachea for from five to ten seconds, at times repeated two or three times, we could produce interstitial emphysema. The sequence of events was as follows. With the introduction of air under increased pressure, the respiration became shallow and in some cases stopped entirely, to be resumed again from one to five minutes later. With the sudden blast, pneumothorax was produced and the diaphragm, which bulged downward, caused some distention of the abdomen. As more air was blown in, the abdomen became distended considerably more, and the walls became drumlike on account of the pneumoperitoneum. As the air finds its way through the inguinal rings and the cervical fascial planes, it escapes to the surface and extends to the neck, the floor of the mouth, the face and the retro-orbital space, causing an exophthalmos. Very little air reaches the upper extremities. From below through the inguinal rings the air finds its way through the

2. Hedblom, C. A.; Joannides, Minas; and Rosenthal, S.: Pulmonary Abscess; An Experimental Study, Ann. Surg. **88**:823 (Nov.) 1928. Joannides, Minas: The Etiology of Pulmonary Abscess, Surg. Gynec. Obst. **47**:449, 1928.

fascial planes upward to the costal margin and spreads posteriorly to the opposite side of the abdominal wall. Anteriorly the air does not spread beyond the midline because the fascia is fixed, but it can travel posteriorly as no fixed points are present. If the emphysema is not marked, the dog may remain in this condition for several hours and then recover so that in from two to ten days no evidence of air is present. Most animals, however, died and at autopsy we found an acute tension pneumothorax with compression of the lungs. We also noted air bubbles at the hilus of the lungs at the point at which the parietal pleura joins the visceral pleura. It appears that this junction has a loose union with the lung, and air can easily escape at this point when the lung is injured. Bullae of air of varying sizes were noted in the lung. No doubt when the intrapulmonic pressure becomes increased, the pleura gives way and the air balloons up this loose portion of the pleura. In a study of the histology of the lung in the living dog, we noted areas in which there was an oblique or sagittal section of the smaller bronchus,[3] the inner half of which subdivided into its normal divisions but the outer half remained open and was covered only by the thin loose pleura. With each inspiration this area distended slightly more than the rest of the surface, and under increased intrapulmonic pressure the loose pleura separated more and a bulla of air was formed. It is ruptured bullae such as this, in addition to leakage of air from the mediastinal areolar tissue, that produce acute pneumothorax. The air bubbles in the mediastinum extend upward toward the cervical fascia and downward through the postesophageal areolar tissue to the mesentery and retroperitoneal tissues. At the hiatus esophagus of the diaphragm an anatomic study revealed the presence of a small inverted triangle, the sides of which were joined together by means of thin fibrous tissue.[4] Under increased pressure from above, the air finds its way through this opening into the abdominal retroperitoneal tissues and spreads along the root of the mesentery downward to the fascia of the iliopsoas muscle and out through the inguinal ring to the superficial tissues in the abdominal wall. At the region of the perirenal areolar tissues frequently a bulla will form which in turn ruptures and allows the air to escape into the free peritoneal cavity thus causing a pneumoperitoneum. When the air travels upward, it passes through the cervical planes and reaches the subcutaneous tissues of the chest. When the sublingual tissues

3. Joannides, Minas, and Olkon, D. M.: The Structure of the Alveolus Pulmonalis in the Living Dog: A Capillaroscopic Study, to be published. Joannides, Minas: Surface Stereomicroscopy of the Lung in the Living Animals, to be published.

4. Joannides, Minas: The Relation of the Hiatus Esophagus of the Diaphragm to the Stomach, Arch. Int. Med. **43**:61 (Jan.) 1929; The Influence of the Diaphragm on the Esophagus and the Stomach, ibid. **44**:856 (Dec.) 1929.

became distended with air, the tongue was pushed up from its bed, thus making it impossible for the dog to breathe. With an additional amount of air under pressure, the face swelled up, and finally the retrobulbar tissues of the orbit caused a marked exophthalmos. Examination of the heart and the general circulation revealed the presence of air in the blood stream. When the heart was opened, we found that in all the chambers the blood changed into a foamy mass, and air bubbles of varying sizes could be traced in the arterial as well as in the venous tree. Capillaroscopic studies by Dr. Olkon revealed the presence of microscopic air bubbles in capillaries of the heart muscle, the kidneys, the bladder, the stomach, the intestines, the brain and the skin.

The results of these experiments necessitated the explanation of the mechanics of several factors. 1. What causes the emphysema? Does it necessarily depend on the increased intrapulmonic pressure per se, or is it secondary to a tear in the lung tissue. 2. What causes death in these animals? Is it the acute tension pneumothorax, the air embolism, the pneumoperitoneum or the interstitial emphysema per se? 3. In the dogs that survive the shock of the experiment, what happens to the air in the tissues and the circulation? 4. How can these animals be saved after the syndrome develops? Would the animals survive if an outlet was made for the air through the skin, the thoracic (to relieve the pneumothorax) or the abdominal wall (to relieve the pneumoperitoneum)?

To determine the effect of the presence of air in the subcutaneous tissues, air under pressure was injected subcutaneously by means of a trocar. In this series, the dog was merely ballooned up and showed evidence of discomfort owing to the distention of the skin. The air was injected at the right inguinal region, but promptly traveled upward and spread throughout the whole body except the extremities. As soon as the trocar was removed, the air began to leak out through the opening made by the trocar. An attempt to suture that opening caused the air to leak out through the new openings made with the needle. The more incisions present in the skin, the quicker the air escapes from the interstitial tissues. If the animal is left alone the ballooning disappears, but crepitation remains and persists for from a week to ten days. Such an experiment was not sufficiently shocking to the animal to cause death. When the animal was killed, we found no alterations in the deeper structures.

The presence of an acute tension pneumothorax, alone or combined with pneumoperitoneum, was then studied. In the series of dogs, the air was injected into the abdominal cavity under sufficient pressure to cause distention of the abdominal wall. In this series the air did not leak out through the inguinal rings although the intraperitoneal pressure was high enough to cause urinary and fecal incontinence. Aside from the discomfort produced by the compression of the intra-abdominal

organs, no other symptoms were present. All these animals survived the shock of the experiment. In another series, an acute tension pneumothorax was produced by the injection of air into the chest through a needle. Depending on the compression of the lung produced by the pneumothorax, varying degrees of respiratory and circulatory embarrassment developed. In marked cases, asphyxia and death occurred; in more moderate cases, dyspnea occurred, and when the abdomen was opened from below, the diaphragm was seen to lose its tone and bulge downward into the abdomen. In this series a mediastinal, retroperitoneal and subcutaneous emphysema developed, but no air embolism was found. It appears, therefore, that an acute pneumothorax plays an etiologic part in the production of interstitial emphysema. Is it, then, the acute pneumothorax that kills these animals? In order to determine this point, we introduced compressed air into the lungs after we had previously inserted a small rubber catheter into each side of the chest to allow the escape of air from the pleural cavity. In this series we noted the characteristic subcutaneous, mediastinal and retroperitoneal emphysema. These dogs, however, tolerated this change after a period of apnea lasting from fifteen to thirty seconds, so that the experiment could be continued for from two to three hours, when ordinarily a few blasts of compressed air into the lungs proved fatal. From these observations we may be justified in assuming that relief from acute pneumothorax keeps the animal alive in the presence of an interstitial emphysema.

The production of air embolism in these experiments has been fairly constant. In more than 80 per cent of the dogs that developed interstitial emphysema by the intratracheal injection of compressed air, we found large amounts of air in the cardiac chambers and in the general circulation. Air was present on the venous as well as on the arterial side, in the systemic as well as in the pulmonary circulation. To determine the effect of air embolism per se we injected air into the femoral vein. When from 200 to 300 cc. of air was injected slowly over a period of from thirty to forty-five minutes, the dog remained alive. If, on the other hand, this amount was injected rapidly over a period of from four to five minutes, the dogs died promptly. Artificial respiration, cardiac massage or intracardiac injections of epinephrine failed to resuscitate the animal. When the heart was incised, we found the blood in a foamy state, and air bubbles of varying sizes were also present. Air could also be traced in the carotids, the aorta and the vena cava. It is possible, then, that with acute interstitial emphysema if the tension pneumothorax does not kill the animals, the air embolism does. But how does the air get into the general circulation under these conditions? There are two possible routes. First, air may reach the circulation by means of a bronchovenous fistula. Such a fistula has been

produced experimentally by C. M. Van Allen [5] with a resultant air embolism. The second route is directly through the capillaries of the lung without any gross injury to the lung tissue. In a stereomicroscopic study of the lung in living animals, such as dogs, rabbits, alligators and frogs, we found that when the animal struggles or there is an increased intrapulmonic pressure microscopic bubbles of air are seen in the larger capillaries, and if such struggles persist these air bubbles become more and more numerous and extend to larger vessels. The frog is the best animal for such a study. We [6] have noted that with a slight struggle air finds its way into the larger vessels without any apparent change in the structure of the alveolar membrane. If the intra-alveolar pressure remains persistently high, all the larger capillaries become devoid of blood cells, and gradually the air bubbles reach the heart cavity. What probably happens is that the alveolar tension is increased to such a degree that more air passes into the capillaries than occurs under normal tension, but after the larger capillaries are reached the tension is again changed to normal with a resulting air embolism.

Another important fact which the studies on the frog revealed is that the alveolar membrane is made up of ridges and grooves, and the blood runs from the many celled capillary at the periphery over the surface of the alveolar membrane and divides into a massive delta so fine that air could easily find its way into these grooves without destruction of the membrane. When air embolism is produced, unless it is of a massive degree, the animal survives. The animal dies as a result of one of two cause: First, as the air reaches the heart chambers it is churned with the blood during the cardiac contraction and the blood becomes foamy. In this physical state of the blood, the contractions of the heart, instead of propelling the blood into the arteries, cause a greater emulsification. The animal dies of asphyxia and anoxemia. The second cause of death is air ambolism of one of the vital centers in the medulla. When air finds its way there, it may remain long enough to cause anemia of any of these centers with possible death.

CONCLUSIONS

Interstitial emphysema is apparently a protective mechanism on the part of the body to keep air from parts where it may cause difficulty. A simple subcutaneous emphysema is harmless, and with a few small multiple incisions the air may be allowed to escape to the outside without causing any harm. If the condition causing the emphysema is persistent and as a result there is an acute pneumothorax and a pneumoperitoneum, the condition may be relieved if an outlet for the air is

5. Van Allen, C. M.: Personal communication to the authors.

6. Joannides (footnote 3, second reference).

produced in the chest and abdominal cavities. No method has as yet been developed, however, to clear up air embolism of a massive type.

We have tried the combination of blood transfusion and venesection in order to replace the emulsified blood with normal blood, but without success. The condition of mild mediastinal emphysema and mild degree of air embolism is probably more common than is generally seen, because heretofore we have recognized only the extreme degrees of the condition. Our experiments lead us to believe that anything that produces an increased intrapulmonic pressure, such as a severe cough, child labor, lifting of heavy objects, etc., probably causes a mild degree of mediastinal emphysema and air embolism, which as yet we have no way of recognizing clinically.

A REVIEW OF UROLOGIC SURGERY

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(Concluded from page 184)

URETER

Tumor.—McCown [30] stated that primary carcinoma is rare, and that the paucity of symptoms makes diagnosis difficult. Early treatment is necessary to secure beneficial results, since tumors of this type result in widespread and rather early metastasis. He reported a case of tumor of the ureter. After various examinations, he concluded that a tumor of the ureter or of the kidney and ureter was present, and retroperitoneal nephrectomy was done through a lumbar incision. On liberation of the adhesions around the pelvis of the kidney, a mass was found in the ureter just below the pelvis, which was about 2 cm. thick. The ureter was liberated and sectioned about 2 cm. above the bladder. Evidence of extension was not found during the operation. Pathologic examination of the specimen revealed papillary carcinoma in the upper third of the ureter, without any evidence of metastasis or implantations along the ureter, as so frequently occurs in papillomatosis of the pelvis of the kidney.

Forty-three cases were reviewed; 21 of the patients were females and 22 were males. The right ureter was involved in 19 cases and the left ureter in 24. The neoplasm occurred in the upper part of the ureter in 5 cases, in the lower part of the ureter in 27 cases, and in the middle part of the ureter in 6 cases; it was diffuse in 1 case, near the center in 1 case, below the center in 1 case, multiple in 1 case, and it

30. McCown, P. E.: Primary Carcinoma of the Ureter, J. A. M. A. **94**:468 (Feb. 15) 1930.

involved the entire ureter in 1 case. The papillary type of carcinoma is the most common. The duration of symptoms from the cases reviewed was from five weeks to fourteen years. Hematuria was the most common symptom; it was noted in 31 cases. Tumor was reported as having been felt in 18 cases; in the final analysis hydronephrosis was reported in 32 cases. The clinical diagnosis of tumor of the ureter was made in 9 of the 43 cases. Nephro-ureterectomy had been performed in most cases. Involvement of the ureteral orifice required removal of a surrounding area of the wall of the bladder.

Stones.—Ibrahim,[31] from his observation in Egypt, expressed the belief that most stones found in the ureter form in the pelvis of the kidney and pass into the ureter. Bilharziasis of the ureter is responsible for the majority of the stones primarily formed in the ureter. In most cases the cause is bilharzial ulceration, infection and surface concretions; in others it is infection of a dilated ureter above a bilharzial stricture. The latter type of stone is usually large and multiple. Most of the stones have a nucleus of oxalic or of uric acid; the remainder of the stone is composed of variable layers of oxalates, urates and phosphates. The variations depend on the chemical composition and reaction of the urine.

Ureteric colic is a constant symptom and is due to overdistention of the urinary tract above the level of the stone rather than to pressure of the stone or to spasm at the point of impaction. Frequency, pain and difficulty of urination occur with stones in the distal end of the ureter, but rarely accompany stones of the proximal end. In cases of intramural stones the picture may closely simulate that of vesical calculus. Hematuria, often only microscopic, is less common than in cases of renal calculus and is more severe during and after an attack of colic. The loin is always tender to deep pressure, as is the course of the ureter, especially at the level of the pelvic brim. The kidney may sometimes be felt enlarged. In a few cases the calculus may be felt on rectal or vaginal examination. Bilharziasis, when present, makes diagnosis more difficult. The urine is usually acid, showing erythrocytes and pus cells on microscopic examination. When complete obstruction occurs, pyuria disappears, although the condition of the patient becomes progressively more serious. When anuria caused by calculus occurs, the other kidney is usually absent or unhealthy.

Many small stones of the ureter pass naturally into the bladder, and slightly larger ones may pass under medical treatment. If the stone is more than 0.5 cm. in diameter, operation is indicated. There is less possibility of a rough or a spiculated stone passing naturally. When dilatation of the renal pelvis as a result of obstruction by stone occurs,

31. Ibrahim, A. B.: Stones of the Ureter, Brit. J. Urol. **1**:396 (Dec.) 1929.

expectant treatment is contraindicated regardless of the nature of the stone. In cases associated with vesical calculus, Ibrahim operates on the bladder several weeks before he operates on the ureter. In cases associated with renal calculus of the same side and of a size small enough to make it possible for its later descent into the ureter, the kidney and ureter are operated on at the same time. If the renal stone is larger, it is removed by a second operation some time after the operation on the ureter. If the condition is bilateral, the better of the two sides is operated on first. In bilateral cases, if the stones are both in the distal end of the ureter, they may be removed advantageously at one operation through a median suprapubic incision. If bilharziasis is associated, the end-results are less satisfactory, but the decision concerning the operation is not affected. In operating on the upper part of the ureter, the ordinary lumbar incision is made because the ureter is not adherent to the peritoneum at this point. In operating on the lower part of the ureter, either the iliac or the median extraperitoneal approach may be used, the iliac being preferable. The incision is started near the anterior superior iliac spine and passes down, parallel to and 2.5 cm. above Poupart's ligament to about 2.5 cm. lateral to the pubic spine. The aponeurosis of the external oblique and the fibers of the internal oblique and transversus abdominis muscles are divided in the line of the incision in the skin. After the fascia transversalis is cut, the deep epigastric artery is divided between ligatures. The peritoneum is reflected to the bifurcation of the common iliac artery, where the ureter is looked for. It is usually retracted with the peritoneum but may be recognized by its feel and by the longitudinal arrangement of its vessels. Not more than 2.5 cm. of the ureter is dissected, care being taken not to denude the periureteric tissue which contains the blood supply of the adjoining segment of the ureter.

In 2 cases of calculous pyonephrosis with multiple small calculi in the kidney, the corresponding ureters being free, nephrectomy was performed. After operation the patients showed marked improvement from pain and intoxication from which they had been suffering. Later, definite signs and symptoms of severe cystitis developed. On cystoscopic examination the orifice of the ureter was found to be inflamed, immobile and patent, with pus exuding from it. The patients in these cases were successfully treated by excision of the remaining stump of the ureter by the suprapubic extraperitoneal route. A small stone was found in each stump. The supposition is that these stones must have slipped down the ureter from the kidney during manipulation for nephrectomy.

Over a period of three years, there was no mortality in 44 cases of stones of the ureter in which operation had been performed. At Kasr-el-Ainy Hospital, 2 deaths (4.8 per cent) occurred in 42 cases.

Bumpus and Thompson [32] summarized the data in 1,001 cases of stone in the ureter, observed over a period of nine years. Pain originating in the renal area and radiating toward the bladder was noted in 634 (63.4 per cent) of the cases, but only rarely did it radiate to the inner side of the thigh or to the genitals. Pain in the lower right quadrant without radiation and with little suggestion of renal colic occurred in 138 cases. Because the symptoms simulated those of appendicitis, the appendix was removed in 37 (26.8 per cent) of the cases, without relief from symptoms. Of the 1,001 cases, appendectomy had been performed in 226. Four hundred fifty-six patients noted marked frequency during attacks and 254 did not have urinary disturbances. Hematuria noticeable to the patient occurred in 300 cases and was demonstrated microscopically in 57 cases. Anuria was the most alarming symptom, occurring in 27 cases; in 22 it was apparently reflex in type, since obstruction was not demonstrable in the opposite ureter. The reflex anuria did not last more than twenty-four hours in any case. Anuria of longer duration is probably due to obstruction.

Stone could not be demonstrated by roentgenograms in 21 (2.1 per cent) of the cases. Without cystoscopic and other data, the definite diagnosis of stone in the ureter cannot be expected in such a high percentage of cases, because many of the shadows are indistinguishable from phleboliths. In 554 cases there was definite obstruction to ureteral catheterization. In many cases, this was passed easily with a catheter, usually with little, if any, obstruction to the passage of urine.

In 146 cases the stone was passed following the manipulation incident to the first cystoscopic examination, whereas in 274 other cases manipulation was followed by success in 202 (74 per cent); in 65 of these, ureteral meatotomy was done as an aid to manipulation, with scissors or with fulguration. In 63 cases surgical removal was necessary after the failure of manipulation. Two of these patients, after their condition had been rendered critical, consented to operation but both died from sepsis. There were 252 cases in which stone was removed surgically from the lower third of the ureter and 228 in which the upper and middle thirds of the ureter were involved. In 49 cases the obstruction produced by stone in the lower part of the ureter had resulted in such extreme ureteral and renal injury that nephrectomy or nephro-ureterectomy was required. Excluding these 49 cases, 60.7 per cent of all the stones in the lower part of the ureter were removed by manipulation. The operations performed in 529 cases were: ureterolithotomy, 372; nephro-ureterectomy, 51; nephrectomy, 37; ureterectomy (nephrectomy elsewhere), 3, and combined operations, 66.

32. Bumpus, H. C., and Thompson, G. J.: Stones in the Ureter, Surg. Gynec. Obst. **50**:106 (Jan.) 1930.

Attempts to deliver stones by cystoscopic manipulation should not be carried to a point at which there is danger of suppurative pyelonephritis. Bumpus and Thompson are of the belief that in cases which show marked infection treatment should be by ureterolithotomy rather than by manipulation; the same holds true in cases with stones which are more than from 1.5 to 2 cm. in diameter and which are known to have been present for a considerable period.

Reaction following manipulation may be minimized if ureteral catheters are left in the ureter to insure drainage following the removal of the stone. The edema produced by the manipulation, together with the resulting ureteritis and periureteritis unless adequate drainage is insured, results in rapid ascending infection. When this has occurred, delay in operating to relieve the stasis and infection greatly increases the risk. In all the cases in this series in which operation was performed as soon as signs of renal infection appeared, the patients recovered. The 2 deaths from sepsis gave a mortality in the series of 0.2 per cent following catheter manipulation. There were 9 deaths following the surgical treatment of ureteral stones. The majority of these cases were complicated by the presence of renal stones and poor renal function, and the operation was done as a life-saving measure. The mortality in the entire series of 1,001 cases was 1.1 per cent.

[Ed. Note.—These authors summarized from a large series of cases what would seem a proper evaluation of the various methods of treatment in cases of ureteral stones. The high incidence of previous appendectomy in the patients comprising this group is noteworthy and is a reflection on the failure of the average general surgeon to employ urologic methods of investigation in the usual case of so-called chronic appendicitis. If the roentgen rays and cystoscope were used more frequently, or even as a routine measure in such cases, many patients would be saved the ordeal and expense of an unnecessary laparotomy.

We agree with the contention that when catheter manipulation is followed by sepsis or when impassable obstruction is encountered, then the indications for prompt surgical intervention are imminent.]

Retained Stone After Nephrectomy.—Hunt [33] stated that subsequent operations on the ureter after removal of its adjacent kidney occur infrequently. Operations are seldom required on the ureter after nephrectomy has been performed for extensive infections of the kidney. Although infection of the ureter is often associated with infections of the kidney, patency of the ureter provides drainage of infectious material from the ureter with subsequent resolution of the process. If this were not true, ureterectomy would frequently be indicated after nephrectomy.

33. Hunt, V. C.: The Necessity for Operations on the Ureter, Including Ureterectomy, Subsequent to Nephrectomy, J. Urol. **23**:43 (Jan.) 1930.

At the Mayo Clinic, during the last ten years, renal tuberculosis has provided the indication for nephrectomy in approximately 28 per cent of the major lesions of the kidney for which this operation has been necessary. In no instance in the series of approximately 574 cases in which nephrectomy was performed for renal tuberculosis during this period has subsequent ureterectomy been recorded. Experience has shown that the ureter, like the bladder, rarely remains infected after a tuberculous kidney has been removed. A search of the records at the Mayo Clinic revealed only one instance in which ureterectomy was necessary after nephrectomy for pyonephrosis. Noncalcareous infections of the ureter have been demonstrated to be rarely the cause of persistent pyuria subsequent to nephrectomy. The most common cause of pyuria from a ureter on the side on which the kidney has been removed is lithiasis, and if pyuria is present the indications are clear for ureterolithotomy or, preferably, ureterectomy.

Foreign Bodies.—Stevens [34] stated that foreign bodies in the ureter, with the exception of stones, are rarely found. Entrance into the ureter may be directly from outside the body or from the kidney, intestines or bladder, the latter route being the most common. Contraction of the bladder or regurgitation may force or carry a foreign body from the bladder into the ureter. It may then travel upward as high as the pelvis of the kidney as a result of ureteral antiperistalsis and regurgitation. Catheters, bougies and other instruments should be carefully examined for imperfections before they are inserted into the ureter. Stevens reported two cases of broken instruments in the ureter, one in which a segment of a ureteral catheter, and the other in which a filiform tip was broken off into the ureter.

[Ed. Note.—The dangers of broken off bits of catheters and bougies being retained in the renal pelvis or ureter was well emphasized by Stevens. The care that should be taken in selecting perfect instruments of this type for ureteral insertion and, what is more important, the need for routine observation of the catheter after removal, are obvious. When such an accident occurs the patient should be hospitalized and the urine carefully observed for the missing part. Further ureteral dilatation or even operation may become necessary if the retained particle fails to pass spontaneously.]

Ureterocele.—O'Conor [35] stated that in order to avoid erroneous conclusions, ureterocele must be clearly distinguished from ureteral prolapse. In his series of 19 cases the ages varied from 19 to 60 years;

34. Stevens, W. E.: Foreign Bodies in the Ureter: Report of Cases, California & West. Med. **32**:104 (Feb.) 1930.

35. O'Conor, V. J.: Ureterocele: A Clinical Study of Nineteen Cases, J. Urol. **23**:33 (Jan.) 1930.

there were 12 women and 7 men. The left side was affected in 10 cases, the right in 4, and in 5 the condition was bilateral. General clinical observations support the premise that narrowing of the ostium is the primary factor in its production. The symptoms are variable and depend largely on the character and extent of the lesion in the upper part of the urinary tract. In treating the condition, transurethral or suprapubic coagulation of the cystic protrusion is most generally employed. In unusual cases, especially if the ureterocele contains calculi, suprapubic excision may be advisable. Nephrectomy should be reserved for cases in which injury to the corresponding kidney has been irreparable. When the condition is bilateral, coagulation of both sides should not be carried out simultaneously. In these instances dilatation of the ostium or its enlargement by slitting has been attempted before proceeding with unilateral coagulation. In addition, ureteral dilatation was done in all cases until a no. 12 French catheter could be inserted; this procedure was accompanied by appropriate treatment of the infection of the upper part of the urinary tract.

Hydro-ureter.—Gruber,[36] in a series of experiments carried out on dogs, observed that excision of the ureterovesical valve, the vesical portion of the ureter, leads to the production of hydro-ureter. Removal of from two thirds to three fourths of the intravesical ureter does not render it incompetent to normal intravesical pressure changes. Progressive dilatation did not appear to change the activity of the ureter; both peristaltic and antiperistaltic contractions were observed. The maximal pressure of the ureter during contraction was 28 mm. of mercury. The operation of meatotomy in human beings, performed through the operative cystoscope, is probably a safe procedure, as it appears unlikely that the entire valve will be cut during its performance. In the study of hydro-ureter in two pigs obstruction was not noted, and the valves were found to be edematous and incompetent.

Indwelling Ureteral Catheters in Urinary Surgery.—Gutierrez[37] is of the belief that the indwelling ureteral catheter is the most valuable adjunct in urologic surgery, provided always that a correct diagnosis is made before establishing treatment. In acute clinical conditions, as in calculous anuria, if the indwelling catheter fails to relieve the symptoms, surgical intervention should not be delayed. One of the most common incidents in nephrolithiasis or ureterolithiasis is the calculous anuria or suppression of urine which tends toward the development of uronephrosis and fatal uremia. It has been proved that early operative

36. Gruber, C. M.: The Function of the Uretero-Vesical Valve and the Experimental Production of Hydroureters Without Obstruction, J. Urol. **23**:161 (Feb.) 1930.

37. Gutierrez, Robert: The Value of Indwelling Ureteral Catheters in Urinary Surgery, Surg. Gynec. Obst. **50**:441 (Feb.) 1930.

intervention gives better results. Watson and Cunningham in an analysis of 205 cases of calculous anuria, in which a group of 110 patients was treated expectantly, reported deaths in 80 cases, a mortality of 72.7 per cent, and in another group of 95 cases, in which operation was performed, reported deaths in only 44 cases (46.3 per cent).

The diagnosis must be accurate and definitely clear because of the contraindications to the use of the indwelling ureteral catheter in extrarenal or intrarenal conditions when the pathologic process has become well advanced or does not communicate with the excretory apparatus, as in perinephritic abscess, cortical abscess of the kidney, or well advanced renal tuberculosis or hypernephroma. Surgical intervention is the most imperative hope of cure in the latter cases. The catheter to be used for diagnosis is preferably a no. 6 French, and for routine treatment a no. 7 or 8 French, of the type used in urography because it is more durable and flexible, and produces less discomfort. It is essential that the catheter secure drainage, relieve pain and correct infection. The fixed catheter should be irrigated at least three times daily with a mild antiseptic solution. In certain cases, when possible, two catheters in the same ureter and continuous irrigation may be definitely indicated. The catheter should be left fixed in place until the symptoms are completely relieved.

During operation the fixed ureteral catheter affords ready exposure of the ureter and facilitates any surgical procedure on the pedicle of the kidney. After operation it will serve to secure drainage and prompt healing of the wound, without leakage of urine or the formation of permanent fistula. It will also serve to divert the urine from the bladder, particularly in operations on vesicovaginal fistulas, thus permitting the bladder to heal without infection from the urine.

[Ed. Note.—The use of the indwelling catheter as a means of securing renal and ureteral drainage in various forms of infection and obstruction is well established. A few observers have offered a possible objection on the grounds that the catheter may produce pressure necrosis of the mucous and submucous tissues of the ureter with consequent sloughing. In rare instances death has been attributed to this source. Likewise it is well known that the presence of the catheter in certain acute cases seems to aggravate rather than to ameliorate the condition. Nevertheless, if the indwelling catheter can be kept freely draining, if it is well tolerated by the patient from the standpoint of pain, and if there are no evidences of exacerbation of symptoms, then the placing of such a drainage tube in the urinary tract is undoubtedly a great factor in reducing infection and in saving kidneys that otherwise might have to be removed.]

BLADDER

Surgery.—Keyes [38] stressed the importance of preventing pelvic cellulitis by prevesical section as a preliminary to suprapubic operation on the bladder. The mortality from preliminary suprapubic cystotomy has been estimated at 10 per cent. At Bellevue Hospital, over a period of six years, 102 suprapubic cystotomies were performed preliminary to prostatectomy; 32 of the patients died. In another series of 62 patients on whom a similar operation had been performed by various surgeons, 9 (14 per cent) died. Pelvic cellulitis was the predominant cause of death. By prevesical section, pelvic cellulitis may be prevented. As the first step of the procedure a catheter is tied into the urethra, and bilateral vasotomy and prevesical section are performed. The procedure is as follows: The prevesical space is opened through a median line incision and the distended bladder is recognized by palpation and by the veins visible beneath the posterior layer of the transversalis fascia. This fascia is split approximately in the median line and retracted laterally with the underlying veins. The muscle of the bladder is thus exposed from the upper edge of the pubes to the insertion of the urachus and laterally for about a finger's breadth from the median line. Three stout, plain catgut sutures are then introduced into the wall of the bladder, care being taken not to perforate the mucosa. One suture is inserted into the base of the urachus at the upper end of the denuded portion of the wall of the bladder, and by it the bladder is tied to the sheath of the rectus. Two other sutures are inserted into the wall, one on each side, at the edge of the denuded area, and are left long. A gauze pad, wet with 1:1,000 solution of acriflavine, is then placed against the denuded portion of the muscle of the bladder in the triangular space between the three sutures. The abdominal wall is closed in the usual manner above the point where the bladder is affixed to it. Below this point the muscle is pulled together by one or two sutures so as to prevent its spreading, and a corner of a gauze pad is pulled up through the incision in the skin, which is sutured around it. This drainage is necessary as much serum is thrown out into the wound. The bladder is not opened at this time. The urethral catheter supplies drainage and decompression.

After the operation, low decompression is employed. If no complications ensue, prostatectomy is performed on the third or fourth day. If the urethra will not tolerate the indwelling catheter, or if the patient is delirious and pulls the catheter out, or if his condition is such as to require prolonged drainage before the prostate gland is removed,

38. Keyes, E. L.: Pelvic Cellulitis Following Suprapubic Cystotomy and Its Prevention by Prevesical Section, J. Urol. **23**:119 (Jan.) 1930.

the bladder may be drained suprapubically at any time after twenty-four hours have elapsed from the prevesical section, with the assurance that pelvic cellulitis will not follow.

Suprapubic prostatectomy with the use of prevesical section has been performed on 29 patients without mortality. Such preliminary section is desirable not only because it eliminates postoperative mortality but also because it does away with the interval of waiting for the patient to recover from pelvic cellulitis before prostatectomy can be done.

Suprapubic lithotomy for a large stone is attended by high mortality, which has been attributed to the poor renal function shown by these patients. Preliminary treatment does not affect the renal function nor the infection of the bladder; death occurs from pelvic cellulitis. Transperitoneal cystotomy has been suggested as the safest route of approach because it eliminates the risk of such cellulitis. Keyes has performed this transperitoneal operation successfully on several patients who would doubtless have not survived the suprapubic section.

Tumor.—Kidd [39] stated that the many attempts to perform complete cystectomy for carcinoma of the base of the bladder have not been encouraging because of the high immediate mortality due to shock, sepsis and chronic uremia. The condition of the few patients who have survived the operation has not always been satisfactory. The operation has been most successful when the ureters were transplanted to the skin and the bladder was removed at a second operation. The operation has been attended by the highest mortality when attempts have been made to divert the ureters into the rectum and remove the bladder either at the same operation, which entails considerable shock, or at a second operation, which entails increased difficulties from adhesions caused by the first operation. Kidd performs cystectomy for carcinoma of the base of the bladder with resection of both ureters, and yet leaves the patient with a functioning bladder. In some of these cases there is usually present on one lateral wall and toward the apex of the bladder a healthy portion of mucous membrane. This is sacrificed in the usual operations for total cystectomy, but by Kidd's technic it is used as a graft or plastic flap to make a new bladder, into which the resected ureters can be made to empty their effluents. This operation has been carried out in two cases with success and it is hoped that it may eliminate the dangers and disadvantages of the older methods of cystectomy.

In one case, at operation, the growth was cut away quickly, and no attention was given to bleeding points, which were dealt with at a later

39. Kidd, Frank: Cystectomy for Carcinoma of the Bladder-base with Restoration of Bladder Function, Brit. J. Urol. **1**:380 (Dec.)) 1929.

stage. There remained a healthy portion of the right lateral and superior wall of the bladder supplied with blood by the intact right vascular pedicle of the bladder and still attached to the right lateral wall of the pelvis, and the two ureters cut across and lying loose on the pelvic floor. The bladder was reconstructed as follows: Into each ureter was inserted a special implantation catheter devised for use in resection and transplantation of the ureters into either the bladder or bowel or onto the skin. Two ligatures of catgut were then tied tightly around the ureter. By this device the ureteral catheters were fixed firmly by the ligatures into the mouths of the ureters, yet the urine was in no way prevented by the ligatures from escaping freely from the mouths of the catheters. The ligatures sealed the lymphatics of the ureters, thus closing the path of ascending infection during the process of healing. The ligatures were left long and used at a later stage for fixing the ureters where it is desired to do so. The two ureteral catheters were pulled out through the urethra and left emerging from the penis. The two ureters were left high and dry running across the floor of the pelvis, and their ends were approximated to the severed posterior end of the urethra by means of the catgut ligatures that fixed them to the ureteral catheters. With the help of several catgut stitches the front portion of the right lateral wall of the bladder was drawn inward beneath the two ureters and attached to the tissues on the floor of the pelvis to the left of the rectum. It was then possible to draw the hinder or upper portion of the flap of mucous membrane of the bladder underneath the ureters to the left side of the pelvis and bring it around on the left side and stitch it loosely by two catgut stitches to the upper anterior portion of the right lateral wall of the bladder. In this way the ureters and the tube were more or less surrounded by a plastic flap of mucous membrane. The operation was performed in one hour and fifty minutes.

The patient recovered from the shock of the operation. He had a certain amount of fever from the seventh to the thirteenth day caused by stitch abscesses in the upper portion of the wound; during the remainder of the illness there was no increase in temperature. The ureteral catheters drained clear urine, free from pus, and were taken out on the nineteenth day. A suprapubic tube could be inserted through the suprapubic sinus into the new bladder and could be left in place. Lavage was carried out through the urethral catheter and suprapubic tube for a week. On the twenty-ninth day the tube and catheter were removed, and the bladder was washed out daily with silver nitrate. On the thirty-third day the patient began to urinate naturally and easily. On the forty-third day, the suprapubic wound was healed and the catheter was removed. From then on the patient passed urine naturally and easily through the penis. He had complete continence and could

retain urine for two hours; as much as 120 cc. passed at one time with a good stream. Five months later he was able to retain urine from two to three hours; there was a good stream, no incontinence and no hematuria. Only a trace of pus was found in the urine. There was no thickening of the base of the bladder to be felt by rectal examination.

MacKenzie [40] reviewed from the literature 21 cases of small round cell sarcoma of the bladder to which he added a case of his own, making a total of 22. A marked difference in the age incidence was noted in the two sexes. In 9 of 20 cases in which the site of the tumor was mentioned it was situated around the trigone on the base of the bladder and around the vesical neck. Six were on the lateral walls, extending down to, and in some cases involving, a ureteral orifice, and 4 were on the roof or posterior wall of the bladder. In 7 of the 22 cases, operative treatment had not been given, or it was not stated. In 5 cases palliative cystotomy (3 suprapubic and 2 perineal), and in 1 case palliative ureterostomy was performed; in 1 case in which diagnosis was made at cystoscopic examination, a specimen was removed for examination. In 1 case the tumor was removed through the vagina and radium was applied. In the other 7 cases a radical attempt was made to remove the growth. The results in all cases were extremely poor. Only 1 patient was cured. This patient is alive and well nine years later. In many of the cases, death followed immediately or a few days after the operation. Eight patients died within three months of operation. Two of the patients not operated on lived two and seven years, respectively.

Beer [41] reported 8 cases of total cystectomy and partial prostatectomy for infiltrating carcinoma of the neck of the bladder, with only 1 death (12.5 per cent). In 7 cases in which the operation was done by the extraperitoneal procedure with implantation of the ureter into the skin, there was no mortality. The patient who did not survive died ten days after the operation with infection of the left kidney due to implantation of the ureters into the sigmoid. Of the 7 patients who survived, 1 patient lived five years, 1 lived nine months, 1 with leiomyosarcoma lived two months, and the remaining 4 patients are still alive, four years, one and a half years, seven months, and six months, respectively, after operation. Since all of the patients of this series were males, Beer's technic is applicable to that sex.

Beer concluded that the mortality of extraperitoneal removal of the bladder with the adjacent prostate gland is not prohibitive and the operation can be done in one step with implantation of the ureters into the skin without undue risk to the function of the kidney. Even

40. MacKenzie, D. W.: Small Round-cell Sarcoma of the Bladder, with Review of the Literature, Brit. J. Urol. **1**:359 (Dec.) 1929.

41. Beer, Edwin: Total Cystectomy and Partial Prostatectomy for Infiltrating Carcinoma of the Neck of the Bladder, Ann. Surg. **90**:864 (Nov.) 1929.

though the patients are obliged to wear an apparatus for the collection of urine, they are relieved of their original painful condition, are rendered fairly comfortable and are well able to get about.

[ED. NOTE.—Total cystectomy would seem to offer the best opportunity for cure in cases of widespread carcinoma of the bladder which are irresectable with the bladder in situ. C. H. Mayo and Hunt, stimulated by the work of Coffey, have recently been perfecting the technic of ureteral transplantation into the sigmoid. Their results have been encouraging. If this method of shunting the urinary stream proves practicable, it would seem to offer a better mode of disposing of the output of urine than that of transplanting the ureters into the skin, as practiced by Beer. Regardless of the method of choice for subsequent drainage of the urine, the contention that total radical removal of the bladder and prostate gland for widespread malignancy will be followed by a higher percentage of cures seems well established in the light of early experience with the method.]

Young [42] stated that in radical resection of infiltrating tumors the best results have been obtained at the vertex and adjacent portions of the bladder. In such cases, if the peritoneum overlies the site of the tumor, it has been his practice to open the peritoneal cavity and carry out resection at a point distant from the growth. In this way a sufficiently wide area of peritoneum is removed in one piece with the wall of the bladder. In cases of tumor of the posterior wall of the bladder not involving the vertex, the intraperitoneal operation is often not satisfactory, because of the difficulty of reaching the peritoneum in the deepest part of Douglas' pouch and the bladder below that point.

Young recently employed a new procedure in which the tumor of the posterior wall of the bladder with its peritoneal coat is resected entirely through an intravesical operation. The technic is as follows: A long median-like incision is made from the pubes almost to the umbilicus and recti muscles separated in the median line and widely retracted. Incision is made into the bladder in the median line, rather close to the prostate gland, and carefully enlarged above and below and widely retracted until a good view of the interior of the bladder is obtained. The ureters are then located and their proximity to the tumor and its infiltrated base determined. The area of removal is demarcated by placing eight Allis clamps in cardinal positions around the tumor, and at a distance of at least 1.5 to 2 cm. from its outermost invasion. Making traction on the upper clamp, a transverse incision is made with the scalpel through the mucosa, submucosa and muscle of the bladder until

42. Young, H. H.: Treatment of Certain Vesical Neoplasms by Intravesical Resection of the Entire Bladder Wall with the Peritoneal Coat, J. Urol. **23**:269 (Feb.) 1930.

the outer surface of the peritoneum is recognized. This is then picked up with two Allis clamps and divided transversely with a knife. The resection is carried out by scissors on each side, going well away from the mass and external to the Allis clamps. When the lower portion to be resected is reached, if it is found to extend beyond Douglas' pouch, the peritoneum is divided transversely at the depths of the culdesac, and resection continued on downward and across the lowermost area selected for resection. Thus the mass, with its peritoneal covering is removed in one piece.

By such exposure it is possible to include retrovesical and periureteral structures in a more radical manner than when complete mobilization of the posterior and lateral walls of the bladder and the delivery of the bladder externally has been carried out.

Schüller [43] stated that the efficacy of radiotherapy in urology is not standardized. In his experience, 3 papillomas of the bladder reacted favorably to deep applications, and in 1 case, an apparently recurrent papillary carcinoma of the trigone disappeared after the insertion of radium needles, followed by the use of deep applications. He concluded that papillomas of the bladder are amenable to roentgen treatment, and that such treatment should be tried in cases in which resection is impossible and before ureteral transplantation to the bowel is undertaken.

Extravesical Tumors.—Hochloff [44] reviewed 106 cases of carcinoma of the cervix in which he had made cystoscopic examinations. These were divided into three groups. Group 1 consisted of cases in which the mucous membrane was normal or there was hyperemia of the trigone, folding of the walls and tiny hemorrhages in the mucous membrane. Group 2 consisted of cases in which the cystoscopic picture was bullous edema of the trigonal mucous membrane and other areas of the bladder. The ureteral orifices were usually raised on a swollen background and their contour was changed. Group 3 consisted of cases of diffuse, square-shaped areas of edema of the mucous membrane, especially over the area of the trigone, making it almost impossible to find the ureteral orifices. In other cases the orifices were somewhat tilted and surrounded by an edematous hyperemic wall. Dilatation of the blood vessels in the mucous membrane were characteristic of the cases in groups 2 and 3.

In group 1 no difficulties were found at operation in the parametral tissues. In group 2 difficulty not infrequently arose in freeing the ureter. The uterus was only slightly movable and there was infiltration of the adjacent connective tissues. In group 3 usually diffuse spreading of the carcinoma into the wall of the bladder was found.

43. Schüller, Hugo: Zur Radium- und Röntgentherapie in der Urologie, Ztschr. f. urol. Chir. **27**:374 (July) 1929.

44. Hochloff, A. W.: Zur Wertung der Chromocystoskopie beim Carcinoma colli uteri, Ztschr. f. urol. Chir. **27**:438 (July) 1929.

Rupture.—Campbell [45] stated that rupture of the bladder occurs rarely but the condition is diagnosed with difficulty and the mortality, therefore, is high. In many instances the diagnosis is made only at operation or at necropsy. Of 55 patients treated in Bellevue Hospital, 35 (63.6 per cent) died, a mortality rate which is in accord with other observations. More than 90 per cent of the ruptures occur in males. Of the patients suffering from intraperitoneal involvement 26 (73.5 per cent) died; only 9 (42.9 per cent) of those with extraperitoneal involvement died. The operative mortality of the group with extraperitoneal involvement was 40 per cent; of the group with intraperitoneal involvement, 68 per cent. A summary of reports of cases from the literature indicated that from 60 to 80 per cent of patients die even under the most favorable surgical conditions.

Distention and alcoholism and other forms of mental irresponsibility are important predisposing causes of vesical rupture. In 19 cases, the ruptures occurred during acute alcoholism. In 9 cases (16 per cent), the rupture was due to injury by automobiles; 8 of these patients were pedestrians. In 11 cases, the rupture followed a fall. Apparently the distance fallen is comparatively unimportant; 1 patient who died fell only 4 feet. Another, who lived, fell a distance of eight floors. In 12 cases, the rupture resulted from crushing injuries. Associated pelvic fracture occurred in 20 cases. In 166 cases of fracture of the pelvis occurring over a period of nine years, there was vesical rupture also in 25 (15 per cent). In 3 others, rupture of the posterior urethra had occurred.

In most cases of rupture of the bladder, shock follows the injury; one fourth of the 55 patients were in shock, were comatose, or were moribund. The general appearance and physical condition of many of these patients strongly indicated internal hemorrhage. Involvement of the urinary tract is suggested by local tenderness of the bladder, dysuria, hematuria or absolute inability to urinate. If intraperitoneal rupture has occurred, early symptoms of peritonitis may be present. Abdominal tenderness and rigidity appear first, then nausea and vomiting. Extraperitoneal rupture was found in 21 cases, whereas intraperitoneal rupture with free urine in the abdominal cavity was observed in 34. Marked abdominal rigidity was present in 22 cases, and unusual distention was seen in 31.

The emergency condition of most of these patients precludes protracted diagnostic procedures. Catheterization is the most commonly employed and one of the most inaccurate diagnostic tests. Often there is no return of urine, or a small amount of blood may be withdrawn. At

45. Campbell, M. F.: Rupture of the Bladder: A Clinical Study of Fifty-Five Cases, Surg. Gynec. Obst. **49**:540 (Oct.) 1929.

times a large amount of urine will flow, more than the apparently undistended bladder would indicate, which signifies drainage of an abdominal cavity filled with urine. The injection of a measured amount of fluid and the estimation of its return should always be attempted, although this method is not absolutely accurate. If the catheter has penetrated an abdominal pool of urine, a larger quantity of fluid will be withdrawn. Diagnostically, this constitutes the most significant of any of these observations. Catheterization, although used in 41 cases, was of diagnostic value in only 13. In almost all cases, urine containing blood was withdrawn. When physical conditions permit, cystoscopic examination is the most accurate method of diagnosis.

Operation should be performed as soon as the diagnosis of ruptured bladder is made. Anesthesia by nitrous oxide and oxygen or by ethylene is preferable in most instances. Spinal anesthesia is contraindicated in the presence of marked vascular depression with low blood pressure. In most cases intraperitoneal exploration is indicated. Operative speed is essential. If no evidence of urine within the peritoneum is found the peritoneum is closed and the interior of the bladder is investigated. If peritoneal involvement is present, such fluid as is readily obtained is aspirated and ample drains are left in place. Flushing out the abdominal cavity, with solution of sodium chloride or medicated solutions, is inadvisable as it wastes time and disseminates infection. The vesicoperitoneal tear is hastily closed by a minimum of sutures firmly grasping the musculature of the bladder. It is unnecessary to attempt to suture the layer of muscles of the bladder. Liberal suprapubic drainage of the bladder is of prime importance. Campbell uses a three-fourth inch tube for this purpose. With free drainage, bleeding and extravasation will stop at once and smaller unsutured bladder wounds will heal readily. Penetrating wounds caused by the bony spicules of pelvic fracture are rarely large enough to require suture. Bony spicules, stones or other foreign bodies in the bladder should be removed while it is open.

Of 20 patients who were operated on and survived, 5 were able to leave the hospital in less than three weeks; 6 left in from three to six weeks; 5, in from six to twelve weeks, and 4 remained in the hospital more than three months. The longest stay was six months. Of the 35 patients who died (10 of whom were not operated on), 2 lived less than ten minutes after admission to hospital. Ten died within the first twenty-four hours; 14 died within three days, and all but 4 died in less than one week. One lived eight months. Campbell concluded that if the patient survives the first week, a fair prognosis is likely. In all cases a grave prognosis is warranted during the first week.

[Ed. Note.—Since rupture of the bladder is a condition which the average urologist encounters infrequently, this report from a clinic in which a large volume of work is done on patients suffering from trauma

is of more than passing interest. Campbell emphasized mental incompetence, distention of the bladder and a variable type of trauma as the factors usually associated in producing the condition. The urgent need for immediate diagnosis as well as the difficulties encountered in making oneself certain of the existence of the lesion are well outlined. The necessity for prompt surgical intervention in the intraperitoneal types of rupture and the early avoidance of operation in vesicorectal and vesicovaginal ruptures are in keeping with good judgment according to Campbell's experience.]

Submucous Cystitis.—Donohue [46] stated that there are two separate and distinct etiologic factors concerned in the production of submucous cystitis: extension from long-continued or recurring infections of the mucosa of the bladder, and dissemination from inflammatory foci in structures outside the urinary tract. Lesions representing extensions from infections within the bladder are usually extensive, the condition of contracted bladder being the common sequel. When submucous cystitis is the result of embolic hematogenous bacterial invasion, the lesions are usually well localized, the remainder of the bladder retaining its elasticity and tonus. Beneficial results cannot be assured in treating extensive lesions of either type of submucous cystitis. Lesions which are not extensive and well localized regularly respond to appropriate treatment.

[Ed. Note.—Donohue differentiates submucous cystitis as due to long-continued or recurring infection of the bladder or due to focal infection, according to the concept of Hunner. Whether the former should be included as true submucous cystitis was challenged during the discussion of this paper. The main point stressed by Donohue and with which urologists of experience are in accord is the deplorable inadequacy of present methods of treatment, such as resection, fulguration, and overdistention. No one suffers more or can be offered less than a patient afflicted with this disease.]

Perforation.—Wilhelm [47] stated that perforation of the bladder during cystoscopic examination occurs more frequently than the few reports in the literature of a similar condition would indicate. Cystoscopic examination should not be attempted in the case of unruly and uncooperative patients, and it is hazardous to perform it under gas and oxygen anesthesia. The introduction of fluids or gases into a perforated bladder for diagnostic purposes is a dangerous and unreliable procedure. Diagnosis of this condition should be made by means of the clinical history. The differential diagnosis of intraperitoneal and extraperitoneal perfora-

46. Donohue, P. F.: Submucous Cystitis, J. Urol. **22**:465 (Nov.) 1929.

47. Wilhelm, S. F.: Perforation of the Bladder During Cystoscopic Examination, J. Urol. **22**:555 (Nov.) 1929.

tion is difficult, if not impossible, and in cases of doubt exploratory laparotomy should be performed. When the perforation is into the rectum or vagina, the treatment is nonoperative. Immediate surgical intervention is indicated in all other cases of perforation of the bladder.

PROSTATE

Hypertrophy.—Takeichi [48] found that the conservative use of radium in the treatment of prostatic hypertrophy affords good results. Improvement in urination and a definite receding in the prostatic swelling were demonstrated. Complications were not observed during the course of the healing processes.

Carcinoma.—From the reports of the Necker Clinic, Dossot [49] studied 154 cases of carcinoma of the prostate gland, including 82 histologic examinations of the operative specimen and 63 reports on necropsies. Prostatic adenoma plays an important part in the pathogenesis of numerous carcinomas; in 11.6 per cent of cases malignant transformation was noted. Two types of prostatic carcinoma should be distinguished: urethroprostatic adenocarcinoma which develops from the adenomatous glands of the prostatic urethra, and true carcinoma of the prostate gland which develops from the gland itself. Among 134 cases of primary carcinoma there were 61 urethroprostatic adenocarcinomas, 17 adenomas suspected of degeneration, 46 true carcinomas of the prostate gland, and 6 probable true carcinomas with adenoma. In 13 cases the nature of the carcinoma was not specified. Carcinoma and adenoma were found associated in 58.7 per cent of cases.

Geraghty, in a series of 450 prostatic carcinomas, noted its association with hypertrophy in 75 per cent of cases, but it is his belief that the two conditions are absolutely independent. Shaw, in more than 1,000 operative specimens of carcinoma of the prostate gland examined at Johns Hopkins Hospital, found only 2 cases in which the carcinoma developed in the middle of the hypertrophied lobule.

Extension in carcinoma of the prostate gland occurs early, and before it can be recognized clinically the carcinoma has extended far beyond the limits of the gland and has invaded the pelvic and abdominal lymph nodes.

Prostatectomy and irradiation have given only mediocre results, and cures of more than three years are unusual. True cures can only be obtained in cases in which operation is performed with a diagnosis of adenoma and in which histologic examination shows the existence of

48. Takeichi, T.: Erfahrungen mit der Radiumtherapie bei Prostatahypertrophie, Japan Ztschr. f. Dermat. u. Urol. **29**:31 (Jan.) 1929.

49. Dossot, Raymond: Cancer of the Prostate: Its Origin and Extension, J. Urol. **23**:217 (Feb.) 1930.

carcinoma. When carcinoma has progressed to a recognizable stage, it is generally extensive. With the present state of knowledge of carcinoma of the prostate gland it is advisable to limit efforts to palliative treatment, such as the passage of sounds and cystostomy.

TESTIS AND EPIDIDYMIS

Seminal Vesiculectomy.—Gutierrez [50] reviewed the results of operation on 100 mentally defective patients for definite pathologic lesions of the seminal vesicles. The operations had been performed six years previously. In 98 cases, bilateral seminal vesiculectomy was performed and in the other 2, vesiculotomy and prostatotomy. Ether and gas were used for anesthesia. All the patients of this series recovered uneventfully both from the immediate traumatism of the operation and from the general condition. Perineal or rectal fistula has not been seen, nor has epididymitis or any other common urologic complication occurred. The membranous urethra, although exposed during the operation, was not opened in any instance. Deaths did not result from the operation, although there were 8 deaths due to different causes, occurring at an average time of two years and three months after operation. On reexamination of the patients, the ampulla was found to be patent and easily palpable by rectum in more than 21 per cent of the cases. Sexual function was apparently unchanged in most instances. Of the specimens removed at operation, 38 per cent showed infection or positive culture. Histologic sections revealed the presence of inflammatory changes of chronic vesiculitis in many cases. Forty-eight per cent of the patients were dismissed from observation or were definitely improved; 52 per cent remained mentally unimproved. The elimination of foci of infection is especially worthy of consideration in dealing with mentally defective patients.

Scrotal Gangrene.—Gibson [51] reported a case of idiopathic gangrene of the scrotum. The onset was sudden, without apparent cause, and was accompanied by extreme toxemia. The gangrene spread rapidly from the scrotum along superficial planes to the axilla. The patient died four days after onset, presenting the typical picture of a severe case of gas gangrene. Necropsy did not reveal a cause of the disease.

Two hundred six cases were reviewed from the literature. The mortality of the group was 26.7 per cent. The three main characteristics of the symptoms of the disease were the sudden appearance of the phe-

50. Gutierrez, Robert: Later Results of Surgery of the Seminal Vesicles: Report of One Hundred Consecutive Seminal Vesiculectomies, J. A. M. A. **93**: 1944 (Dec. 21) 1929.

51. Gibson, T. E.: Idiopathic Gangrene of the Scrotum with Report of a Case and Review of the Literature, J. Urol. **23**:125 (Jan.) 1930.

nomena in the midst of apparent health, the rapid evolution of mortification, and the apparent total absence of any of the usual causes of gangrene. The process spreads frequently upward from the scrotum along superficial fascial planes and may extend to the axilla. In many cases no external lesion or disease of the genito-urinary organs can be discovered to explain the origin of the infection. Two theories may be considered as to its inception: a slight external scratch or abrasion unknown to the patient at the time serves as a portal of entry for the infecting organisms, or a minute lesion, such as an infected node in the urethra, is the starting point of the infection. The disease is subject to considerable variation in virulence; patients die as early as thirty-six hours and as late as thirty-three days after onset. Gangrene usually appears about three days from the onset of symptoms.

Treatment in general should be similar to that used in cases of extravasation of urine, that is, incision and disinfection. Because of the undoubted anaerobic nature of some, if not all, infections of the external genitals accompanied by gangrene, the use of antigangrenous serum or anaerobic antitoxin should be given a trial. Even though the whole scrotum sloughs away, satisfactory regeneration will take place.

Dislocation of the Testis.—Alyea [52] stated that cryptorchidism occurs in from 0.1 to 0.2 per cent of all young adults. Aberrant migrations of the testis are less common than cryptorchidism. The congenital perineal testis is the most common type; more than 100 cases have been reported.

Twenty-three original cases of traumatic dislocation of the testis were found to be reported in the literature, to which Alyea added 2 cases. The resultant position of the dislocated testis is dependent on anatomic abnormalities, on obstruction to dislocation in particular directions and on the direction and force of the blow. In most cases the etiologic factor was the passage of a wheel over the genital region; this was followed by severe shock, local pain, and nausea and vomiting. Diagnosis was usually simple after the disappearance of traumatic swelling. Treatment was satisfactory; reduction without open operation was accomplished in three cases, and open reduction was performed in 16 cases.

Herniation or compound dislocation of the testis would seem to be a condition likely to occur in many industrial accidents, but only 3 cases are reported. One of the 2 cases presented by Alyea is of this type. Prognosis in all cases is excellent.

Tuberculosis.—Wang [53] stated that it is difficult to evaluate the results of treatment with tuberculin, particularly when it is part of a

52. Alyea, E. P.: Dislocation of the Testis, Surg. Gynec. Obst. **49**:600 (Nov.) 1929.

53. Wang, S. L.: Tuberculin Therapy in Urologic Tuberculosis, J. A. M. A. **94**:235 (Jan. 25) 1930.

general course of treatment. Other factors, such as the natural tendency of most patients to improve after operation and the periods of latency of symptoms which occur with both inoperable and postoperative cases of tuberculosis of the genito-urinary tract must also be considered. In a series of 55 cases of inoperable, postoperative and preoperative tuberculosis in which lesions were localized in the genito-urinary tract, an effort was made to ascertain whether the patients were benefited by tuberculin treatment, according to the plan that has been carried out. Of the 55 patients, the condition of 39 was improved, of 8 it seemed stationary, and of 7 it was retrograde. One patient died. Besides the tuberculin treatment, these patients received mercury-vapor-quartz light and other treatment. It could not be determined which of these procedures was the most favorable, but tuberculin appeared to have been helpful. In the three years during which the tuberculin had been administered it was evident that there were few, if any, standards for its use in this form of tuberculosis. Tuberculin is not a cure for tuberculosis of the genito-urinary tract, as demonstrated by the time and extent of its usage, but there is considerable evidence that it aids in building up the general health of the patients. In the treatment of patients with inoperable conditions following operation, it has seemed to be a helpful factor.

PENIS

Leukoplakia.—Fukai [54] reported on the rare occurrence of leukoplakia or leukokeratosis of the penis. He found reports on only 43 cases in the literature since the first case reported by Perrin in 1892. Recently he had observed 6 such cases. Usually the lesions occur in congenital phimosis. The process begins as a small wartlike structure, but later becomes diffuse and covers the glans and the inner lining of the prepuce. One case progressed to the formation of carcinoma of the urethra.

URETHRA

Bleeding.—Glingar [55] differentiated spontaneous hemorrhage from the meatus. If spontaneous bleeding is from the external meatus, it is definitely an anterior urethral hemorrhage. Hemorrhage during micturition is a form of significant hematuria. It is important to determine whether the blood originates from the urethra and from which specific portion. Initial hematuria signifies urethral bleeding without definite localization; terminal hematuria means bleeding above the external sphincter, although, under some conditions, bleeding from the

54. Fukai, A.: Leukokeratosis glandis et praeputii penis, Acta dermat. **11**: 528 (June) 1928.

55. Glingar, A.: Pathologie und Therapie der Harnröhrenblutungen, Ztschr. f. Urol. **23**:81, 1929.

anterior urethra may simulate terminal bleeding, whereas total hematuria leaves all possibilities open. For topographic diagnosis endoscopic, urethroscopic and cystoscopic examinations are necessary. Ejaculatory bleeding is one of the causes of hemospermia. Experience has shown that bleeding associated with defecation occurs in the posterior part of the urethra.

URINARY ANTISEPTICS

Kaufman [56] stated that in the treatment of infection of the urinary tract the importance of drainage should be recognized. Water is essential, with certain physiologic aids, in combating pyrexia, toxemia and renal failure.

The purpose of urinary antiseptics is clearly established on both scientific and clinical grounds in the treatment of acute and chronic infections. Clinical cure is obtained in acute pyelonephritis and cystitis by alternate alkalinization and treatment by acid hexamethylenamine. This treatment does not usually bring about actual sterilization of the urinary tract. In chronic urinary infections, including those without serious organic lesions and those in which infection persists after operative removal of organic lesions, urinary antiseptics are promising aids. No single antiseptic has been found which is universal.

Hexamethylenamine is of definite value, particularly as a prophylactic against infection in instrumentation of the bladder, in the simpler forms of acute infections, and in general routine postoperative care. Kaufman expressed the belief that acidifiers should always be used and found that a safe combination was phenyl salicylate, methenamine and sodium benzoate. Intravenous therapy offers an opportunity for prompt concentration of the antiseptic. Mercurochrome-220 soluble, if used intravenously in effective dosage, produces severe reactions. For local use, mercurochrome is the most effective for actual sterilization, especially in operative cases. For general use, acriflavine was observed to be better tolerated although apparently less efficient. It is Kaufman's belief that mercurochrome is superior to acriflavine in the treatment of staphylococcus infections, as well as in chronic infections from colon bacillus. Silver nitrate is invaluable in actively stimulating repair, a factor which may materially assist in sterilization of the urinary tract. Boric acid is a passive antiseptic, indispensable for cleansing; it does not irritate and is clinically satisfactory.

Thomas and Wang [57] studied six compounds, none of which could be considered as an ideal urinary antiseptic. When mercurochrome is

56. Kaufman, L. R.: A Clinical Consideration of Urinary Antiseptics, J. Urol. **22**:163 (Aug.) 1929.

57. Thomas, B. A., and Wang, I. K.: Studies on the Comparative Clinical Values of Various So-Called Urinary Antiseptics, J. Urol. **22**:22 (July) 1929.

given in dosage of 300 mg. three times daily by mouth in salol-coated pills "about 30 per cent of the urine from the bladder will become antiseptic." Hexylresorcinol administered in 25 per cent olive oil in dosage of 0.6 Gm. three times a day has the same disadvantages as mercurochrome, and the antiseptic value in the urine is much less. Fifteen grains (0.97 Gm.) of hexamethylenamine three times daily frequently caused indigestion, and the bactericidal effect on the urine was not certain. The germicidal strength of methylene blue was higher than expected, but the dye did not usually produce sufficient concentration in the urine to be of antiseptic value. Pyridium administered in dosage of 0.2 Gm. three times daily by mouth proved to be a weak antiseptic, and its action against the colon bacillus was practically valueless. Salol has no standing as an urinary antiseptic, since the phenol content excreted in the urine never reaches a germicidal strength. It apparently rendered the urine bland and less irritating to an inflamed urinary tract, thereby giving a certain amount of comfort to patients.

[Ed. Note.—One must conclude from this and from other recent studies of the comparative value of urinary antiseptics that they are still far from ideal. No drug is as yet available which will render the urine strictly bactericidal in vivo. We are forced to the conclusion that in chronic infections of the urinary tract we must consistently rely on the methods of instrumental drainage and lavage as a means of therapeutic attack.]

UROLOGIC CONDITION IN EARLY LIFE

Stevens [58] reviewed the histories of the last 9,115 patients admitted to the pediatric department of Stanford University Hospital and found that 205 suffered from one or more pathologic conditions of the urinary tract. One hundred six of the 205 patients were males and 99 females. Pyelitis occurred in 74 cases, nephritis in 35, phimosis in 28, undescended testis in 13, cystitis in 11, enuresis in 10, pyonephrosis in 6, urethral stricture in 5, nephrolithiasis in 3, redundant prepuce in 3, hydroceles in 4, and tuberculosis of the kidney in 2. In infancy and childhood pyelitis is the most common pathologic condition of the urinary tract. It occurred in almost a third of the patients and was found about four and a half times as often in females as in males.

Urinary calculi are frequently observed during infancy and childhood. They are found more often in the lower part of the urinary tract, although they usually originate in the kidney. In 2,313 reported cases of pathologic conditions of the urinary tract, renal calculi were found in 11 cases, ureteral calculi in 4 cases, vesical calculi in 7 cases, and urethral calculi in 1 case. Although stricture of the urethra follow-

58. Stevens, W. E.: Diseases of the Urinary Tract During Infancy and Childhood, J. Urol. **23**:61 (Jan.) 1930.

ing gonorrhea is rare in children, congenital strictures or stenoses in both sexes are not uncommon. They occur usually at the external meatus.

The same urologic procedures that are used in adults are indicated for the diagnosis and treatment of diseases of the urinary tract in infants and children. Postponement of treatment often results in permanent injury to the kidneys or other urinary organs. The general practitioner, pediatrician and urologist should cooperate to the utmost in the investigation and treatment of pathologic conditions of the urinary tract during infancy and childhood.

ENURESIS

Horton [59] reviewed the subject of enuresis and its treatment, particularly with reference to cases occurring in families compelled by economic circumstances to live in small crowded homes, where the problem presents, in addition to clinical aspects, a sociologic side. Five per cent of the total number of children attending the Children's Out-Patient Section of the London Hospital in one year suffered from enuresis.

In a study of 60 cases an endeavor was made to discover any common factor in its incidence and to attempt to obtain a quicker response to treatment by simple psychologic methods in conjunction with the use of belladonna. There were 23 boys and 37 girls in the series; 56 per cent of these were aged between 5 and 8 years on their first examination. Fifteen per cent of the children were aged more than 9 years, and 28.4 per cent were between 3 and 5 years. In 41.6 per cent of the cases enuresis dated from infancy and in 57.4 per cent the onset occurred after control of urination had been established over varying periods, from one month to seven years. A family history of enuresis was given in 56.6 per cent of the cases; in two cases enuresis had been present in the grandparents.

Various diseases occurred directly antecedent to the onset of the trouble. A rheumatic history was given in 13 per cent of the cases. Six cases dated from an attack of measles, and 4 originated in severe frights. The symptoms were anorexia, restless sleep, insomnia, night terrors, constipation and headache. A complete general examination did not reveal abnormality in 48.3 per cent of the cases; there were varying degrees of general debility in the majority of these. Enlarged tonsils were found in 23.3 per cent and thread worm in 8.3 per cent; cervical adenitis occurred in a smaller proportion. Phimosis was present in only 1 case. Urinalysis was negative in the majority of cases.

59. Horton, Kathleen M.: Enuresis in Hospital Practice, Arch. Dis. Childhood 4:105 (April) 1929.

Patients with incontinence by day constituted only 5 per cent of the total group. Thirty per cent of the patients had enuresis only at night. It was about equally distributed between boys and girls. Enuresis was present both day and night in 55 per cent of the series. In 10 per cent of the cases incontinence of feces was associated with enuresis.

The following plan of treatment was carried out: regular weekly attendance for a considerable time and regular administration of the medicine. The child is not to be worried, scolded or punished. Tea, coffee, cocoa, soups, broths and meat extracts are not given. No more than four small cups of milk, milk and water or water alone are to be taken in the twenty-four hours. No drink is taken after 5 o'clock in the evening; slices of lemon or orange may be sucked for thirst. Salt fish or highly seasoned food are avoided, as well as heavy puddings and much potato. The child is put to bed early, on his side, and tight bedclothes and a hard mattress are used. Urination should be at regular intervals and at the same time each day. The physician's share in the treatment consists in the correction of any local irritation, for example that due to urine, constipation, thread worms and drugs. The number of dry days and nights for each week are recorded, and unlimited opportunity for voluntary micturition during school hours is offered. Intake of fluid is increased gradually after complete freedom from enuresis, has been obtained for a month. Tincture of belladonna is given, beginning with 5 minims (0.3 cc.) three times a day and increasing by weekly increments of 5 minims up to 30 or 35 minims (1.9 or 2.2 cc.), with ammonium bromide 5 grains (0.32 Gm.) three times a day, increasing by 1 or 2 grains (0.065 or 0.13 Gm.) each week up to 12 grains (0.78 Gm.). Tincture of nux vomica in doses of 2 or 3 minims (0.12 or 0.18 cc.) is sometimes added to the mixture. The dose of the belladonna and bromide mixture necessary to help the child to secure seven dry days and seven dry nights is maintained for a fortnight and then reduced by the same amounts as it had been increased.

The results obtained by this treatment were very satisfactory. Sixty-five per cent of the patients were cured, whereas the remaining 35 per cent all showed improvement. The diurnal trouble cleared up six weeks from beginning of treatment. The nocturnal trouble cleared up in the same length of time in half of the cases; the remaining half was composed of cases in which attendance was not satisfactory. Complete recovery from both day and night enuresis was sometimes delayed as long as the eighteenth week.